DISEASES *of the* CHEST

VOLUME XII MARCH-APRIL, 1946 NUMBER 2

Acute Primary Diaphragmitis* (Hedblom's Syndrome)

MINAS JOANNIDES, M.D., F.C.C.P.**
Chicago, Illinois

DEFINITION

Hedblom's syndrome is an acute primary myositis of the diaphragm. Its clinical manifestations are inspiratory pain (ponopnea) on the affected side, limitation of mobility of the lower chest wall, and a tendency to a flaring out of the costal margin with each inspiration. There may also be pain in the upper quadrant of the abdomen with a tendency to muscle spasm. The patient may complain of pain in the homolateral shoulder and the trapezius ridge. Roentgenologically there is a tendency of the involved diaphragmatic leaf to rise and also have a limited or even absent mobility. As the course of the disease progresses on to healing, the diaphragmatic mobility is resumed but the normal contour of the diaphragm now changes so that the dome of the diaphragm may be flattened out.

HISTORY

There have been references in the literature in regard to the possibility of this condition. Most references presume that such a lesion is secondary to pathology of the pleura and the lung from above and have generally referred to it as diaphragmatic pleurisy. Other references presume that such a lesion may be associated with lesions in the subphrenic area with irritation of the peritoneal surface of the diaphragm.[1] We prefer to refer to these lesions as secondary diaphragmitis in contradistinction to Hedblom's syndrome which is a primary lesion in the muscle fibers

*Read before the Southern Chapter of the American College of Chest Physicians, St. Louis, Missouri, November 14, 1944.

**From the Department of Surgery, College of Medicine, University of Illinois, Chicago, Illinois.

Copyright, 1946, by the American College of Chest Physicians.

of the diaphragm with extension of the pathology to the pleura above and the sub-phrenic area below.

This lesion was first described by us[2] in 1935, four years after we saw the first patient with this lesion. The condition was named Hedblom's syndrome in honor of the late Dr. Carl A. Hedblom, our former chief, a pioneer in thoracic surgery, who inspired us with enthusiasm and the desire to carry on in the field of thoracic surgery. By attaching Dr. Hedblom's name to this entity we have been able to attach a more euphonious name than "Joannides syndrome" or even the cacophonic term of acute diaphragmitis. This entity should not be referred to as acute phrenitis because that term designates lesions of the brain.

Osler[3] has come closest to describing this lesion accurately. He states that "from its importance in respiration any disturbance of the function of the diaphragm may result in marked symptoms, especially in respiration. With inflammation of either surface the proper contraction is affected." Here also Osler is referring to lesions in the diaphragm secondary to those in the pleural cavity or the subphrenic area. He further states that "inflammation is common and usually secondary to some process in the thorax or the abdomen. Primary involvement occurs in trichiniasis and some cases of severe scurvy. The lymphatic supply favors infection.... With irritation of the diaphragm or its serous coverings pain may be felt in the shoulder due to stimulation of the sensory terminals of the phrenic nerve. As a result of an acute process changes in the muscle and adhesions are common with resulting restriction of motion. This causes dyspnea and possibly the pain and soreness so common after pleurisy." Although somewhat confusing, the clinical picture described by Osler is quite accurate. He mentions the restricted respiratory movements during the height of the disease. He also refers to the pain in the shoulder, which, however, is not due to the irritation of any sensory fibers in the phrenic nerve but is a referred pain resulting from the rather high origin of the phrenic nerve in the cervical cord.[5]

With as great authorities as Hippocrates and Osler missing out completely on the description of the primary lesions of the diaphragm and referring to them only as secondary to lesions in the thorax and the subphrenic area, it is obvious that attention has been centered primarily on the coverings of the diaphragm and not the muscle itself.

Our attention was first attracted to this condition by the sequence of events in the first three cases described in 1935.[2] By 1938 seventeen cases had been collected in our series. To date forty-two cases have been collected in our own practice. It is interesting to note that in the cases seen by us in consultation,

the attending physician invariably had made a diagnosis of diaphragmatic pleurisy, basal pneumonia, acute cholecystitis, or acute appendicitis. It is for this reason that the present report is made, in order to bring this entity to the attention of the medical fraternity so that patients may have the advantage of a more accurate diagnosis, and be prevented from an unnecessary cholecystectomy or appendectomy.

In order that a more clearcut clinical picture of this lesion may be presented, we wish to repeat the description of the first three reported cases and also to include two more recent cases which are characteristic of the disease.

CASE REPORTS

Case 1: Mr. P. D., a fruit peddler, aged thirty-eight years, was caught in the rain on October 11, 1931, and remained wet for eight hours. By the time he reached home, he had developed a severe chill, a temperature of 103 degrees Fahrenheit and an abdominal pain. The pain was generalized at first, but later became localized in the right side of the abdomen. When seen by us two hours after the onset of the chill, the patient complained of painful breathing (ponopnea) and the only comfort he could have was in a sitting position. At this time the lungs showed no evidence of any involvement. There was a definite muscle spasm in the abdomen but it was generalized to the whole right side. The patient was admitted to Columbus Hospital, where a conservative regime was instituted in view of the absence of vomiting and the presence of a normal leukocytic count. A basal pneumonia of the influenzal type was suspected. Oxygen inhalation was instituted in order to combat anoxia due to the shallow breathing caused by the pain which the patient experienced with each inspiration. Morphine was given to reduce the pain. He was kept in a semi-Fowler position because it gave the greatest comfort. The next day the patient developed a friction rub and subcrepitant rales in the right base. By October 14, three days after onset, the abdominal findings subsided. He showed some tendency to cough but refrained from doing so because of pain on coughing. On the next day the temperature came down to normal and remained at that level until he left the hospital on October 16, 1931, five days after onset. When discharged from the hospital, he still complained of slight pain in the back of his chest on deep inspiration but the lung showed no physical signs of any involvement. The discharge diagnosis was influenzal pneumonia for lack of better diagnosis but the condition was too short-lived for a pneumonia and presented findings quite unusual for an acute appendicitis.

The exact picture of the above syndrome was clearly revealed to us when we saw a patient who died following a perforated peptic ulcer. At autopsy he revealed a massive inflammation of the diaphragm with the formation of thick fibrinous exudate extending to the pleural side. An analysis of certain similarities of the symptom complex in the two cases led us to assume that the diaphragm was really at fault in the first case. Following is a report on the second patient.

Case 2: Mr. G. K., a salesman, aged forty-seven years, enjoyed excellent health with the exception of a right inguinal hernia. The patient

gave no history of previous gastrointestinal trouble. He developed a hernia eight years previously by lifting a heavy suitcase. The company surgeon recommended a truss which the patient wore with resultant comfort for eight years. The present illness started suddenly on the morning of April 3, 1932, with a severe stabbing pain in the abdomen which developed when he attempted to get up from bed. There was nausea but no vomiting, no urinary retention, no hematuria. When seen thirty minutes after the onset of his symptoms, the patient was in agony from the severe pain in the abdomen. He presented a typical Hippocratic facies, with sunken cheeks and eyeballs and beads of cold perspiration on his forehead and temples. The knees were drawn. Even the jarring of the bed aggravated the pain in the abdomen. The abdomen was rigid. The respirations were painful and labored. The pulse rate ranged from 68 to 74 beats per minute. There was a definite tenderness over the right inguinal hernia which was reducible but would not remain reduced because of the marked straining by the patient. The truss caused severe pain when applied and was, therefore, removed. The pain did not subside even with morphine, given in two doses of ¼ grain each at an interval of fifteen minutes. An examination of the chest showed no evidence of a pulmonary or cardiac lesion to account for these symptoms. The abdominal wall showed rigidity which was more marked on the right side. The patient was sent to Columbus Hospital. There was no doubt that he presented an acute surgical condition of the abdomen, that he was in shock, that he had a hernia which might have been strangulated. He also presented a characteristic picture of an upper

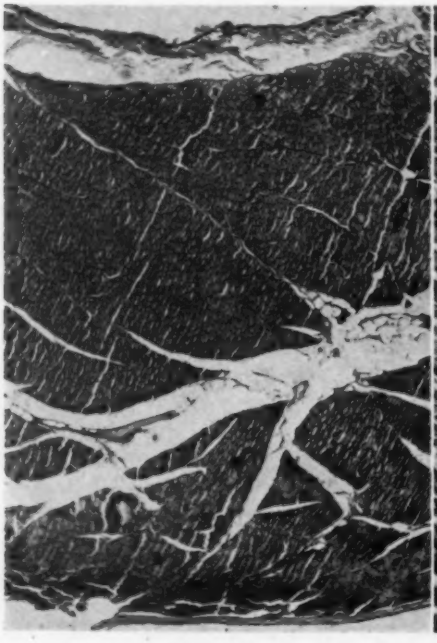

Figure 1

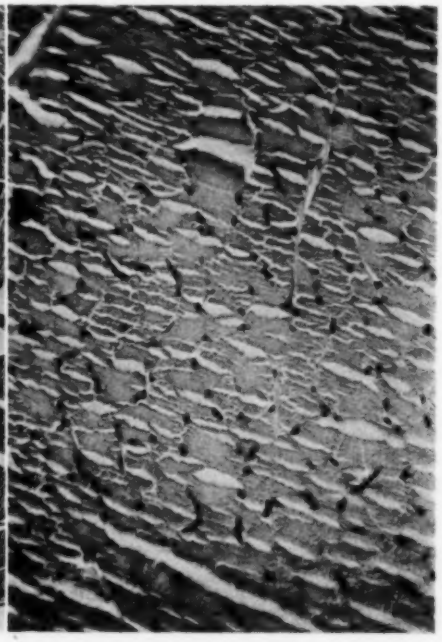

Figure 2

Fig. 1: Cross section of normal diaphragm magnified 85 times. Whole thickness of diaphragm shows in section. Pleural and peritoneal surfaces appear thin and without leucocytic infiltration.—*Fig. 2:* Cross section of normal diaphragm magnified 450 times. Note flattened muscle fibres and absence of leucocytic infiltration.

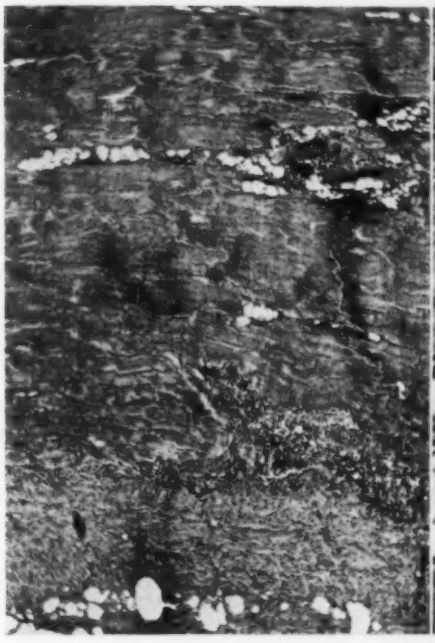

Figure 3

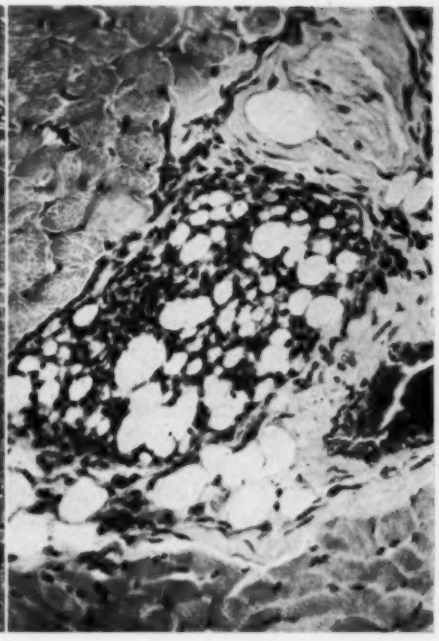

Figure 4

Fig. 3: Acute diaphragmitis. Cross section magnified 85 times. Note swelling of muscle fibres and leucocytic infiltration between the fibres and in the lymph spaces. Pleural surface thickened and unfiltrated.—*Fig. 4:* Acute diaphragmitis. Cross section magnified 450 times. Note leucocytic infiltration in lymph space and swelling of muscle fibres.

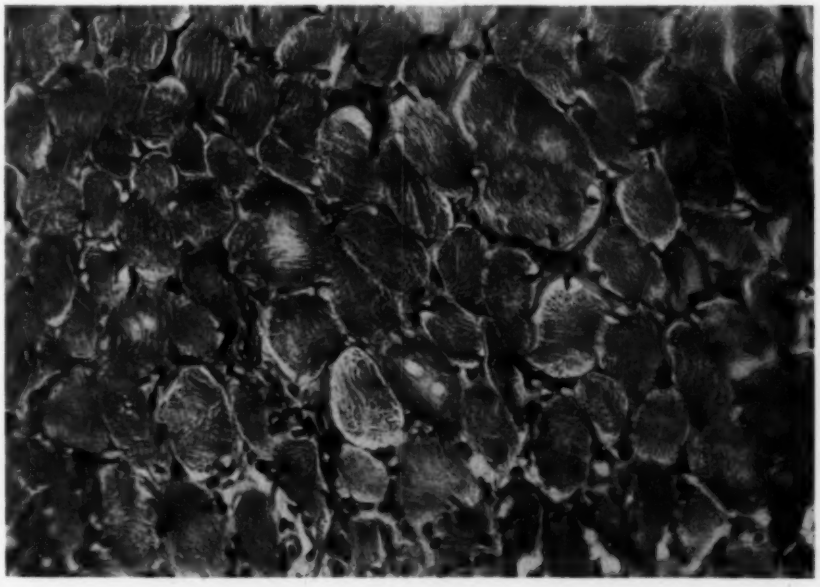

Figure 5: Acute diaphragmitis. Cross section magnified 450 times. Note marked swelling of muscle fibres and leucocytic infiltration between the fibres.

right quadrant surgical lesion, most likely a perforated silent peptic ulcer. An acute pancreatic necrosis was also considered. An x-ray film of the abdomen showed no pneumoperitoneum. On admission the white count was 6,000 but on the following day rose to 30,000 cells per cubic millimeter. The question of surgical intervention was discussed with the patient's wife but she refused to have the patient operated on, therefore a watchful waiting regime was followed. The pain in the abdomen persisted but was slightly reduced by the use of large doses of opiates. The patient had difficulty in breathing because he developed severe pain in the chest with each inspiration. Gradually the respirations became entirely costal in type with definite lagging of the chest wall on the right side and a spasmodic outflaring of the costal margin on the right side with each attempt at inspiration. The patient complained of pain in the right shoulder but this pain was not severe when compared with the chest pains at inspiration and also the abdominal pains. The abdominal wall assumed a board-like rigidity. Examination of the chest on the day of admission showed no pulmonary or cardiac pathology but twenty-four hours later mucous rales were present on both sides anteriorly and in the axillary areas. The facies of agony persisted until the patient went into coma about five hours before death on April 6, 1932, three days after the onset of symptoms. About one hour before death there was a persistent frothing from the mouth, suggesting the development of an acute edema of the lung.

At autopsy, which was done by Dr. Ernest Nora, the lungs showed an acute edema with no free fluid in the pleural cavity. The diaphragmatic surface of the right lung as well as the pleural surface of the diaphragm showed a thick gray exudate with fibrinous adhesions between the lung and the diaphragm. The heart showed no important gross lesions. The abdomen contained some free sero-purulent exudate. The serosal vessels of the whole bowel were injected. No gangrene of the bowel was noted. Above the hepatic flexure 500 cc. of thick grayish-yellow fluid was pocketed between the liver and the diaphragm. The peritoneal surface of the diaphragm showed a thick deposit of grayish-yellow bread-and-butter type of exudate. There was also a prepyloric perforated ulcer located on the side of the lesser curvature. The perforation was the size of a dime. The area around it showed no induration. On section, the diaphragm was three times the thickness of its mate on the left side. The cecum and the site of the hernia showed no important lesions. There were no lesions in the pancreas, thus eliminating the suspicion of a possible hemorrhagic necrosis. Microscopic examination of the diaphragm showed a definite swelling of the muscle fibers with infiltration of polymorphonuclear leukocytes in the lymph spaces and between the muscle fibers (see Figures 1, 2, 3, 4, 5). On the pleural and peritoneal surfaces of the diaphragm the serous coverings were definitely inflamed with a proliferation of the cells and polynuclear infiltration. This case is definitely one of a subphrenic abscess with extension of the inflammatory changes to the diaphragm and through it to the pleura. It is, therefore, a case of secondary diaphragmitis and not one of a Hedblom's syndrome.

An analysis of this case reveals a silent prepyloric ulcer which on perforating caused an acute diaphragmitis, as evidenced by the ponopnea, the immobilization of the chest wall, and the pain in the right shoulder. The local peritonitis was no doubt the cause of the board-like rigidity of the abdomen. It is also possible that the irritation of the diaphragm

may have caused an irritation of the transversus abdominis muscle because at their points of insertion the two muscles interdigitate with one another.[4]

The similarity in the clinical evidence in the two patients, as evidenced by abdominal pain, ponopnea, pain in the shoulder and limited mobility of the chest wall on the affected side, led us to suspect that the primary factor in the clinical picture in the first case was an acute diaphragmitis. That such a syndrome is possible as a primary lesion has been proven to our satisfaction by the third case, in which we have been able to demonstrate roentgenologically an immobilization of the diaphragm, an elevation of the diseased leaf during the acute process, and a flattening out of the dome as the process subsided.

Case 3: Mrs. E. A., a housewife, aged forty, presented herself on July 30, 1934, complaining of headache, nasal discharge and sore throat. On August 9, 1934, she called us to see her at the Garfield Park Hospital, because of pain in the region of the costal margin, at the angle of the scapula and the upper right quadrant of the abdomen. She placed herself under the care of a neighboring physician who made a diagnosis of

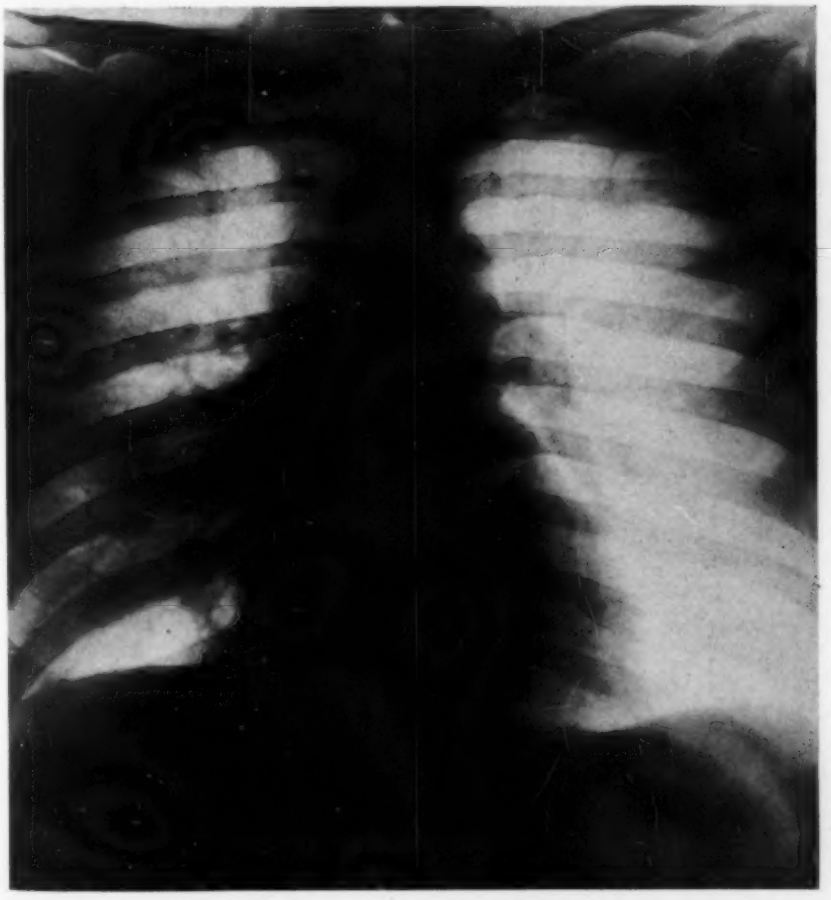

Figure 6: Roentgenogram taken on August 9, 1934. It shows a definite rise of the right diaphragm. There is a slight obliteration of the normal dome of the diaphragm, and also a definite infiltration in the right hilum.

right-sided pneumonia, based on the presence of rales in the chest. When seen by us, she still complained of the characteristic pain in the lower chest and upper abdomen but also had painful breathing. There was little or no cough. Because of our previous experience with the other two cases, we arranged for a fluoroscopic examination which revealed an immobilization of the right leaf of the diaphragm. Roentgen films showed a slight rise of the right hemidiaphragm and a normal dome. There was also some evidence of parahilar infiltration of the lung which was diagnosed as pneumonia. She made an uneventful recovery. She was seen at our office on several occasions after her discharge from the hospital and a serial study of her diaphragm offers us distinct proof that this patient suffered from Hedblom's syndrome. The Roentgen ray studies reveal a moderate rise of the diaphragm on the involved side. There is also a gradual obliteration of the normal dome of the hemidiaphragm, no doubt due to one of two causes. Either fibrous tissue which may have been deposited is beginning to contract, or else costophrenic adhesions are pulling on the muscle and cause the gradual elimination of the normal dome. (See Figures 6, 7, 8, 9).

Case 4: The findings in this case are those of Dr. M. S. of Chicago, who developed Hedblom's syndrome during the postoperative course of an appendectomy to which he submitted. This report was kindly furnished by Dr. M. S. and is published verbatim.

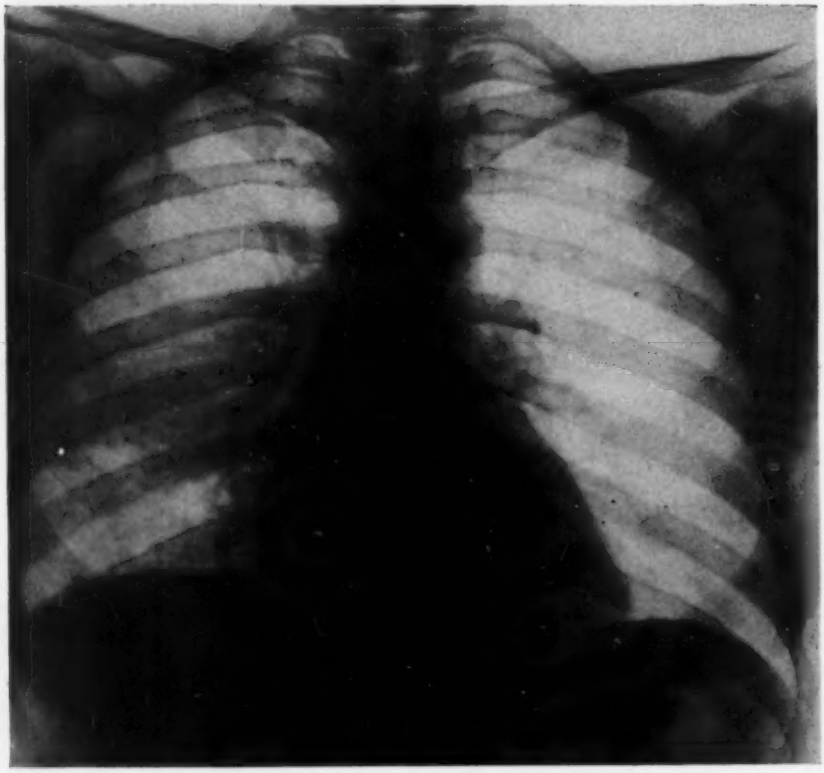

Figure 7: Roentgenogram taken August 14, 1934. The hilar shadow is cleared up. There is a rise of the diaphragm on the right side. The dome of the diaphragm is still evident.

"On January 15, 1939, I was admitted to the Alexian Brothers' Hospital for removal of my appendix which was found to be acutely inflamed. During the convalescent period I was disturbed by severe pain in the right lower quadrant during the ingestion of food. This pain came on with the first swallow of food, but subsided so that I was usually able to finish my meal. I was discharged from the hospital on January 25, 1939.

"These spasmodic pains persisted at home and I soon began to notice a rise in temperature, a few chills and finally persistent pain in the right lower quadrant of the abdomen. I was brought back to the hospital by ambulance on February 1, 1939, and upon admittance my temperature was 102.6, pulse 120 and respirations 20. At this time the chills came on more frequently and were more severe. I was at times delirious. The blood count taken on February 2, 1939, revealed a white count of 30,000. On February 5, 1939, a large amount of drainage with a strong fecal

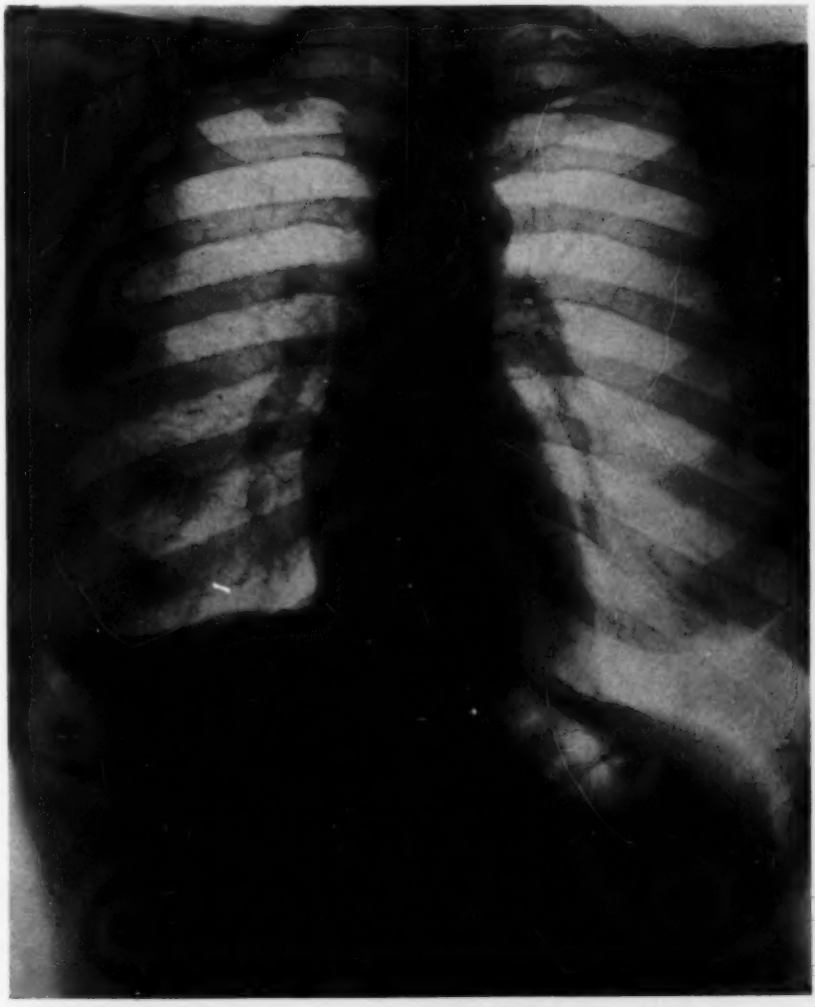

Figure 8: Roentgenogram taken September 17, 1934. The pulmonary markings show no evidence of acute pathology. The dome of the diaphragm is almost entirely obliterated. The costo-phrenic angle is now about 90 degrees. There is a definite rise of the right hemidiaphragm.

odor broke through the operative wound. Following this, I felt better and apparently was on the way to recovery (with the exception of the persistent drainage) until the morning of February 18, 1939, one month after the onset of my appendicitis.

"I awoke that morning feeling well and was up on the back rest for a short time when I felt a severe knife-like pain in the chest on the right side at about the level of the ninth rib in the mid-axillary line. The pain was aggravated on inspiration. There was also a persistent pain in the right shoulder at the trapezius ridge and supraclavicular area. There was also some pain and muscular spasm in the right upper quadrant of the abdomen. At this time I coughed up several mouthfuls of fresh blood and breathing was extremely difficult because of the pain. The pains persisted and were excruciating and there was the desire to cough up. This was done with great difficulty because of the aggravation of pain with each attempt at deep breathing. My temperature rose to 104.4 degrees Fahrenheit, the pulse to 130 and the respirations to 30 per minute. The white count taken the next day was 40,600.

"The presence of blood in the sputum persisted for two months. The pain in the chest likewise persisted even after I was discharged from the hospital. Dry heat and opiates were used to relieve the pain. Calcium gluconate by mouth and Vitamin D were given for the hemoptysis.

"The episode was rather frightening to me and the course rather stormy. However, on March 26, 1939, I was discharged from the hospital,

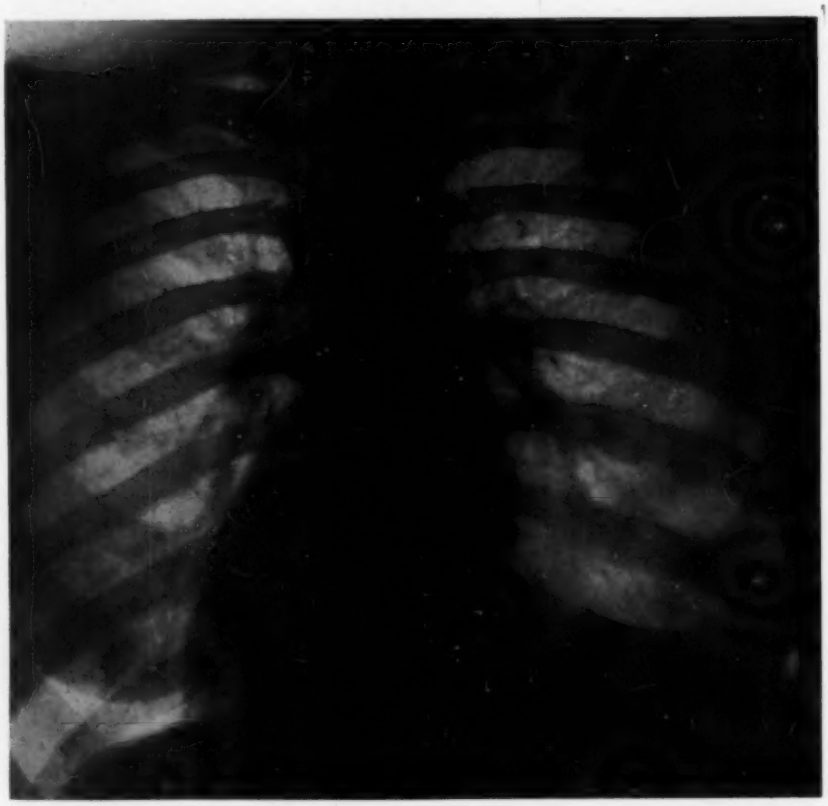

Figure 9: Roentgenogram taken on November 17, 1934. The right hemidiaphragm is still elevated and flat.

at which time my temperature was 98 degrees Fahrenheit, pulse 88 and respirations 22 per minute. Fecal material was still discharging from the appendectomy wound in copious amounts and there were persistent pains in my chest, especially on deep inspiration. I had lost thirty-six pounds in weight.

"The pains in the chest still develop from time to time and are especially aggravated by changes in weather, particularly a sudden drop in temperature. They also develop on exertion five years after the onset.

"I returned to my practice on July 15, 1939."

The findings in this case were those of an acute right-sided Hedblom's syndrome. When seen by us in consultation, the clinical picture of diaphragmitis was obvious and had already progressed to the point of extension of the pathology to the lower lobe of the right lung. It is possible that the findings were of embolic origin with inflammation of the diaphragm and coincident pathology in the lung. The presence of hemoptysis suggested an infarction of the lung which went on to the process of resolution rather than breaking down with a resultant abscess of the lung (see Figures 10, 11). The x-ray findings are characteristic of an acute diaphragmitis with immobilization and rise of the right leaf of the diaphragm and eventual flattening of the normal dome.

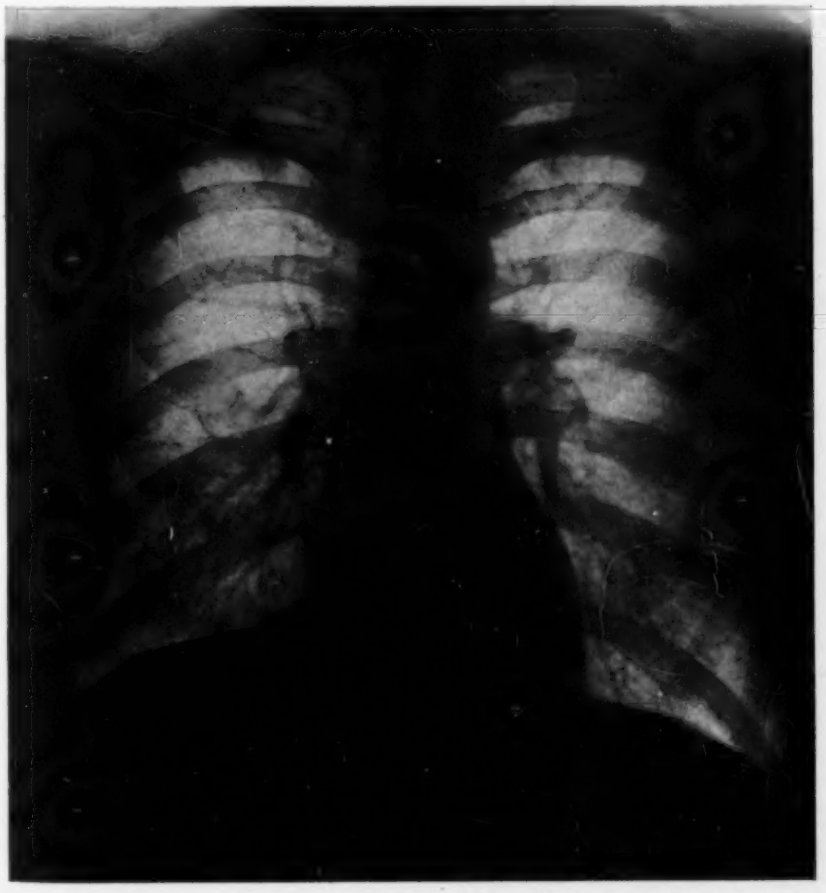

Figure 10: Roentgenogram taken September 4, 1939, seven months after the acute onset. The right hemidiaphragm is still high and flat.

Case 5: This is the most recent case seen to date. Mrs. A. Z. is a patient of Dr. Kurt W. Ossendorff of Chicago, who has kindly furnished us with the clinical history.

Mrs. A. Z., aged sixty-six years, was admitted to St. Joseph's Hospital of Chicago on June 19, 1944. Her chief complaint was pain in the left lower chest which was aggravated on inspiration. The pain was referred to the left shoulder. The patient complained of some cough but she had difficulty in coughing because it aggravated the chest pain. She also complained of pain in the left shoulder and the trapezius ridge. She was seen in consultation on June 28, 1944, at which time she showed a limited mobility of the left chest wall with some outflaring of the left costal margin. There was no spasm of the transversus abdominis muscle. A few sibilant rales were elicited in both lungs, suggesting a tendency to cardiac insufficiency with some passive congestion of the lungs. X-ray films at inspiration and expiration showed an immobile diaphragm on the left side with an elevation of about five centimeters. Because the findings were not very acute, she was given diathermy over

Figure 11: Roentgenogram taken September 22, 1944, five years and seven months after the acute onset. The right hemidiaphragm is still high and shows flattening.

the affected side with complete recovery. Two months later a follow-up x-ray of the chest showed complete return of the diaphragm to its normal level and contour.

This case is typical of the findings in Hedblom's syndrome. It was diagnosed roentgenologically as a case of diaphragmatic pleurisy by the roentgenologist until we pointed out to him the actual condition involved. Dr. Ossendorf was worried about the possibility of a basal pneumonia which, in the presence of cardiac insufficiency, might go badly with the patient. Fortunately she made an uneventful recovery.

In order to elucidate the various aspects of this entity, we shall discuss its pathologic and clinical problems.

PATHOLOGY

Acute inflammation of the diaphragm presents the same steps as acute inflammation in any muscle. There is a swelling of the muscle fibers with the usual changes of leucocytic infiltration into the tissues. The pathology may become limited to the muscle bundles or may extend to the serous surfaces, namely, the pleura on the upper surface, and the peritoneum on the under surface. In one patient the pathology extended into the hepatic tissue, causing an abscess of the liver which ruptured through the diaphragm into the pleural cavity, resulting in an effusion containing characteristic liver tissue.

As the acute inflammation subsides, the muscle fibers that have been destroyed during the acute stages are replaced by fibrous tissue. Under such conditions the diaphragm loses its normal dome-like shape so that on x-ray examination the muscle appears flattened out. If there is any pleural involvement, adhesions may develop between the base of the lung and the upper surface of the diaphragm. If effusion develops along with the pleural involvement, plastic changes occur at the costophrenic and cardiophrenic angles, resulting in definite widening of these acute angles.

CLINICAL PICTURE

Hedblom's Syndrome may have a sudden onset with pain on the involved side and the pain localized over the costal margin and also on the ipsolateral shoulder. There is a characteristic painful inspiration (ponopnea) and it varies, depending on the severity of the inflammation. If the respiration is too painful, one can see on physical examination a definite immobilization of the lower portion of the chest wall, with a sudden jerky spasm noticeable just below the costal margin in the abdominal wall and just above the costal margin in the chest wall. There may or may not be a cough at onset. The shoulder pain on the ipsolateral side is definitely a referred pain and may be distributed at the trapezius ridge, the supraclavicular fossa, at the region of the anterior

scalenus muscle, and also, less frequently, at the infraclavicular region at the middle of the clavicle.[5,6,7]

Hedblom's syndrome may be preceded by a chill twenty-four to forty-eight hours before the onset. There may also be a history of an acute "head cold" preceding the disease. This syndrome may often be confused with acute appendicitis and at operation a normal appendix is found. In such a case, in addition to the characteristic ponopnea, and the pain along the costal margin, and the immobilization of the diaphragm, as seen with a fluorescent Roentgen screen, we are apt to notice a spasm of the transversus abdominis muscle. This may be a reflex spasm or due to an actual extension of the inflammatory changes to the fibers of the transversus muscle. This becomes obvious when one keeps in mind the intimate relation of the fibers of the diaphragm and the transversus adbominis.[4] If the transversus spasm is limited only to the hypochondrium, an acute cholecystitis may be considered. It is easy to eliminate its presence by the characteristic presence of referred pain to the right shoulder, the presence of ponopnea and the immobilization and rise of the diaphragm on x-ray study. We believe that a definite number of patients with these findings are erroneously diagnosed as acute cholecystitis and have their gall bladders removed unnecessarily.

The extension of the pathology from the diaphragm to the serous surfaces will cause appropriate symptoms. This pleural extension may lead only to a few basal adhesions or an acute exudative pleurisy resulting in an empyema. In the less fulminating cases, where only a small amount of exudate has developed, it is absorbed, but the thickened pleura of the costophrenic angle now causes a shadow of a right angle rather than the normal acute costophrenic angle. This change becomes more obvious when muscle fibers of the diaphragm have been destroyed during the acute stages and have been replaced by fibrous tissue, causing a flattening of the normal dome of the diaphragm.

The less acute cases of acute diaphragmitis (Hedblom's syndrome) clear up within a few days, frequently recur with changes of weather, and are particularly noticeable with the onset of damp weather. Subsequent attacks are usually not severe, although in one case the attack resulted in a fulminating acute pleurisy with effusion and acute septicemia which resulted fatally. The usual physical signs of moisture in the base of the lung are noticeable in the cases where there is coincident or secondary pulmonary involvement.

Fluoroscopically one can see a definite immobilization of the involved hemidiaphragm and a definite elevation of the muscle on the involved side. If the patient is followed up by means of

Roentgen studies over a long period of time, one will observe a gradual diminution of the normal dome of the diaphragm, and eventually almost complete flattening of the involved diaphragm. Such flattening occurs as a result of replacement of the normal muscle fibers by fibrous tissue, secondary to the inflammation of the diaphragm.

DIFFERENTIAL DIAGNOSIS

This syndrome must be differentiated from the following conditions, namely, acute spontaneous tension pneumothorax with pressure over the diaphragm; acute surgical conditions in the abdomen with extension of the pathology to the transversus abdominis muscle or the diaphragm; pleurodynia; herpes zoster intercostalis; scalenus syndrome; cervical ribs; artificial or spontaneous pneumoperitoneum; irritation of the phrenic nerve trunk associated with mediastinal pathology; acute spasm of the diaphragm; and, finally, pulmonary atelectasis, either massive, lobar or lobular.

Acute Spontaneous Pneumothorax: Acute, compressive, tension pneumothorax of the ball valve type in which air enters into the pleural cavity, and then is not released back into the bronchus, has often caused symptoms similar to an acute Hedblom's syndrome. The one-way valve rupture of the visceral pleura causes a gradual and persistent increase of the intrapleural pressure. The degree of pressure rise depends entirely on the size of the bronchus associated with pleuropulmonary fistula. If the mediastinum is fixed, the pressure is usually exerted towards the hemidiaphragm on that side. If a relatively large branch of the bronchial tree is exposed to the pleural cavity, the tendency is to develop a relatively higher intrapleural pressure before the air would stop escaping into the pleural cavity. Under such conditions the pressure over the diaphragm causes a symptom complex much similar to an acute primary diaphragmitis (Hedblom's syndrome). The referred shoulder pain on the same side is usually present. Because of the intimate relationship of the points of attachment of the fibers of the diaphragm and the transversus abdominis underneath the costal margin, we expect to find a definite spasm of the transversus abdominis. This frequently is confusing enough to lead to the diagnosis of an acute appendicitis. I have seen two patients who had a laparotomy by someone else, and who, on careful study, showed a spontaneous tension pneumothorax. Both these patients stood the shock of the operation quite well in spite of the diminished respiratory capacity. These two cases emphasize the importance of careful preliminary chest examination, preferably checked with a routine Roentgen ray film of the chest.[12]

It is striking at times to notice that there is only a slight degree of dyspnea, particularly in the cases in which the mediastinum is quite fixed, and the opposite lung functions without much embarrassment.

Acute Surgical Abdomen: An acute surgical abdomen resulting from any lesion that causes a massive peritonitis may be confused with an acute Hedblom's syndrome. In such cases the inflammation of the peritoneum may extend to the peritoneal surface of the diaphragm, and also to the transversus abdominis muscle. There may be a secondary inflammation of the diaphragmatic muscle fibers, thus causing the characteristic picture of acute primary diaphragmitis (Hedblom's syndrome), in addition to the initial lesion. Frequently the abdominal muscles alone are involved, and particularly the transversus abdominis, which is in intimate relationship to the parietal layer of the peritoneum. An irritation of this muscle causes a definite functional change in the peripheral fibers of the diaphragm because these interdigitate at their point of insertion underneath the chest wall. One may, therefore, see any or all the characteristic findings associated with an acute primary diaphragmitis, namely, ponopnea, referred pain in the ipsolateral shoulder, fixation of the lower chest wall and upper abdomen at the site of the costal margin, and an immobilization and rise of the diaphragm seen in x-ray studies.

Basal Pneumonitis: Inflammation of the lower lobe of the lung per se, in our experience, has shown no findings that would be confused with an acute primary diaphragmitis. If, however, the visceral pleura becomes involved, the diaphragmatic pleural layer of necessity may often become involved, with the result that symptoms will develop referable to a lesion in the diaphragm. Under such conditions one may expect a variable degree of exudative or plastic pleuritis which will result in eventual distortion of the normal outline of the diaphragm.

Pleurodynia, Intercostal Herpes Zoster, and Intercostal Neuralgia: We have been told by a number of our colleagues that the symptom complex which we refer to as an acute primary diaphragmitis has been really that of pleurodynia. A careful study of this latter syndrome has shown us that the condition known as pleurodynia primarily involves the intercostal muscles and not the diaphragm. Pleurodynia may often be confused with earlier stages of herpes zoster intercostalis or intercostal neuralgia.[8] In both conditions one notices a constricting pain in the chest which may lead to ponopnea. This pain follows the course of the intercostal muscles or the nerves involved and bears no relation to the diaphragm. Tenderness may often be elicited along the course of the involved intercostal nerve. We have seen two cases of intercostal herpes

zoster in which the patient developed an excruciating pain in the chest following the distribution of the intercostal nerve. The first case, a man of sixty-five years with a moderate coronary sclerosis, was at first treated as a case of angina pectoris, but within a week he developed the characteristic skin pustules on both sides of the chest close to the sternum and just below the nipples. The second case, a woman of fifty-six years, had excruciating pain in the right lower chest. This pain could be relieved only with large doses of morphine and constant application of heat. She also had a severe diabetes mellitus. During the acute attack, repeated careful examinations of the chest revealed no pulmonary or diaphragmatic pathology. In two weeks after the onset of her pain she developed the characteristic skin lesions of herpes zoster. We have not as yet seen a characteristic case of pleurodynia.

Artificial or Spontaneous Pneumoperitoneum: The presence of air in the peritoneal cavity is of definite interest to us. It is harmless when not associated with a perforation of the bowel. It is absorbed rather rapidly at the rate of 80 to 100 cc. per day. The capacity of the peritoneal cavity varies from 3,000 to 6,000, depending on the size of the patient. When the patient is in an erect position, there is a tendency for the air to find its way towards the undersurface of the diaphragm. Under such conditions anything that would cause an increase in the intra-abdominal pressure will result in symptoms suggestive of Hedblom's syndrome. It is interesting to note that in cases of diaphragmatic paralysis following a phrenic nerve section, there is no pain in the shoulder on the paralyzed side, and there is a tendency for the air to be pumped by the good hemidiaphragm towards the undersurface of the paralyzed side.[7] This phenomenon has been utilized by us in inducing a collapse of adherent lung in patients who have contraindications to thoracoplasty.[9] Such patients may be differentiated from Hedblom's syndrome by the absence of a ponopnea, and the characteristic pneumatic pad seen fluoroscopically underneath the diaphragm. The referred pain to the ipsolateral shoulder is definitely a referred pain resulting from pressure on the diaphragm.

Irritation of the Phrenic Nerve Trunk Associated with Mediastinal Pathology: Lesions anywhere along the course of the phrenic nerve, namely, at the site of the scalenus anterior muscle, or in the mediastinum, may cause characteristic referred pain to the ipsolateral shoulder, or the trapezius ridge. Such lesions may be inflammatory resulting in the formation of scar tissue which may compress the nerve trunk. They may also be neoplastic in type, causing either pressure on the nerve or infiltrating the nerve trunk and causing irritative changes. We have seen two such cases

where pressure on the nerve trunk has resulted from scar tissue secondary to a healed tuberculous lymph node of the tracheobronchial glands. These patients complained not only of pain in the shoulder but also of pain in the interscapular level of the spine, and at times in the region of the xiphoid process. One of these patients complained particularly of severe pain at about 3:00 A. M. which lasted until he got up. The pain then became less severe and did not incapacitate him. Sedatives, including opiates, diathermy and a dry climate have been of no help.

Hedblom's syndrome may be differentiated from this condition by the absence of the characteristic ponopnea and the presence of limitation of the diaphragmatic excursion with each inspiration. There is never a complete paralysis of the diaphragm because, if such paralysis occurs, the referred pain disappears. The normal dome of the diaphragm has been persistent in both patients suffering from this syndrome as contrasted with the flattening of the diaphragm associated with acute diaphragmitis. Both patients with this syndrome complained of pain along the third, fourth and fifth intercostal nerve distribution. The second patient also has anginoid attacks due to the myocardial insufficiency.

Scalenus Syndrome and Cervical Ribs: Naffziger[11] has recently emphasized the clinical importance of the "scalenus syndrome." It is characterized by the presence of pain over the shoulder, frequently extending down the arm without any objective evidence of nerve or circulatory disturbance in the extremity. He states that scalenus syndrome is usually associated with a cervical rib. Relief is obtained by means of scaleniotomy. Because of the presence of shoulder pain this syndrome may be confused with that of acute primary diaphragmitis. It is easily differentiated by the fact that the scalenus syndrome has a slow, progressive onset, while the Hedblom's syndrome has an acute onset. Furthermore, the x-ray findings are quite characteristic in both cases. It is of interest to note that not all cervical ribs are associated with symptoms. In the last five years, in a series of 61,423 x-ray films that we examined, we have recognized over fifteen such cases from the films of the chest and on clinical study found all of them to be symptomless.

Acute Spasm of the Diaphragm: We often notice that patients who have a tendency to intermittent claudication also develop attacks of acute spasm of the diaphragm. This spastic contraction causes a severe pain on the affected hemithorax, especially at the costal margin and the upper abdomen. It usually lasts a few moments and is relieved by making the patient take deep long breaths, and often after taking a long deep breath, to increase the intra-abdominal pressure by straining. A few such

efforts and relief follows. Such a spasm is in no way to be confused with Hedblom's syndrome which is due to an acute inflammation of the muscle fibers of the diaphragm.

Pulmonary Atelectasis: This condition is generally encountered postoperatively. It is usually due to an occlusion of the bronchus either during an operation under a general anesthetic or soon after the operation if the patient is kept in one position and if he is not encouraged to cough and take deep breaths.

Clinically it is manifested by a tendency to dyspnea, rapid pulse, flushing of the face and frequently by fever. At times the patient may complain of pains in the chest or the abdomen. X-ray studies of the chest show an area of atelectasis in the lung with some rise of the diaphragm and possible displacement of the mediastinum. The characteristic ponopnea of Hedblom's syndrome, the immobilization of the diaphragm and the referred pain in the shoulder are usually absent in atelectasis of the lung.

COMMENT

Lesions in the region of the diaphragm have in the past been thought of in connection with the organs above or below the diaphragm, or in connection with the serous coverings of the diaphragm. Capps,[6] in a careful study of the intrathoracic and intra-abdominal viscera extending over a period of twenty years, refers to the diaphragm and assumes that the pain originating in the region of the diaphragm arises from the diaphragmatic pleura or the subphrenic peritoneum. He states that "of great clinical importance is the fact that forcible movements of the diaphragm, such as deep breathing, hiccough or cough, markedly exaggerate the pain." He further states that "inflammation of the margin of the diaphragm sets up pain in the hypochondrium, or even in the lower abdomen on the same side; and that inflammation of the central portion of the diaphragm provokes a sharply localized pain in the neck along the trapezius ridge."

Our attention has been called to the work of Capps in our study of artificial pneumoperitoneum as a therapeutic measure in certain types of pulmonary tuberculosis.[10] Taglia and Narrod,[7] in studying such cases after phrenic nerve interruption, have been able to demonstrate a definite referred pain in the shoulder on the non-paralyzed side, and a complete absence of pain on the paralyzed side of the diaphragm. This pain was relieved when a patient with pneumoperitoneum was lying down and the air found its way underneath the abdominal wall. If, on the other hand, the patient was placed in a sitting position, or if, for any reason, the intra-abdominal pressure was increased enough to produce pressure underneath the diaphragm, the patient complained of

pain in the shoulder much in the same manner as when the phrenic nerve itself is intentionally pinched for localization purposes preliminary to a phrenic neurectomy. Such a finding can, therefore, be used to determine whether the phrenic nerve has been injured. In our experience with over fifteen hundred phrenic nerve interruptions, a large percentage of patients show accessory branches arising from the medial aspects (the sympathetic trunk) and also from the lateral aspect (probably coming from lower segments of the cervical cord).

The severity of the symptoms is variable. In some cases the symptoms were noticed only after a flattening of the diaphragm was first noticed in the x-ray. In such cases a history of repeated mild attacks, often referred to as "pleurisy," is quite common. In the more acute cases a diagnosis of basal pneumonia or upper abdominal lesions have often been looked for. It is obvious that cases of "basal pneumonia" or "pleurisy," or cases with upper abdominal pathology, should be x-rayed and preferably fluoroscoped to study the condition of the diaphragm. Furthermore, the characteristic ponopnea is present in Hedblom's syndrome and absent in upper abdominal pathology. Referred pain in the ipsolateral shoulder should aid considerably in the diagnosis. The immobilization of the lower chest and the upper abdomen, with the characteristic prominence of the costal margin, is quite significant in these cases.

TREATMENT

Our treatment in these cases has been primarily symptomatic. We were anxious to determine the natural course of the disease. Whenever complications arose we took care of them accordingly. We have tried to keep the patients comfortable with sedatives or physiotherapy. The disease itself is self-limited, but has a tendency to recur. The severity of the subsequent attacks is variable. These attacks often follow a cold that comes on with atmospheric changes. The preceding head cold is usually very mild as compared to the severity of the syndrome itself. At times opiates had to be administered to keep the patient comfortable.

CONCLUSION

We are quite convinced that Hedblom's syndrome is a common entity, and has been wrongly labeled under various conditions which are secondary to or associated with it. A more careful clinical study of cases presenting ponopnea and lower chest immobility and particularly upper abdominal pains without other confirming signs of abdominal pathology, will aid materially in the diagnosis of Hedblom's syndrome.

We attach the name of Dr. Carl A. Hedblom to this syndrome in his honor and as an expression of respect and gratitude for the aid and encouragement he had given us as the head of the Surgical Department of the College of Medicine of the University of Illinois. We sincerely hope that he does not resent this posthumous liberty we have taken in identifying his name with this entity.

CONCLUSION

Estamos absolutamente convencidos de que el síndrome de Hedblom es una entidad patológica común a la que se le ha dado equivocadamente el nombre de otros estados secundarios al síndrome o asociados con él. El estudio clínico más cuidadoso de los casos que presentan respiración dolorosa e inmovilidad de la zona torácica inferior y, particularmente, dolores en la región abdominal superior sin otros signos confirmantes de afección abdominal, ayudaría notablemente a diagnosticar el síndrome de Hedblom.

Le damos a este síndrome el nombre del Dr. Carl A. Hedblom en honor a su memoria y como expresión de respeto y gratitud por la ayuda y el estímulo que nos dio como Jefe del Departamento Quirúrgico de la Escuela de Medicina de la Universidad de Illinois. Esperamos sinceramente que no le ofenda la libertad póstuma que nos hemos tomado al identificar su nombre con esta entidad patológica.

REFERENCES

1 "Complete Works of Hippocrates," Foes Edition 1657, P. 18. "The Genuine Works of Hippocrates," F. Adams' Translation, William Wood and Company, New York, 1929, 145.
2 Joannides, M.: "Acute Primary Diaphragmitis (Hedblom's Syndrome)," *American Journal of the Medical Sciences,* 189: 566, 1935.
"Acute Diaphragmitis (Hedblom's Syndrome), a Report of a Study of Seventeen Cases," Publicado en el Tomo II del Libro de Oro Dedicado al Prof. Dr. Mariano R. Castex, Buenos Aires, 1938.
3 Osler, W.: "The Principles and Practice of Medicine," D. Appleton and Company, New York, 1925, pp. 687-688.
4 Joannides, M.: "An Extraperitoneal Transdiaphragmatic Route for Lower Thoracic Surgery," *Ann. Surg.,* 84: 337, 1926.
"An Extraperitoneal Intrapleural Route of Approach for Intrathoracic Surgery," *Ibid.,* 80: 908, 1924.
5 Kuntz, A.: "The Autonomic Nervous System," Lea and Febiger, 1934 Edition, p. 201.
6 Capps, J. A. and Coleman, G. H.: "An Experimental and Clinical Study of Pain in the Pleura, Pericardium and Peritoneum," MacMillan Company, New York, 1932 Edition.
7 Taglia, V. and Narrod, S.: "Diagnosis of Diaphragmatic Paralysis or Irritation" (Personal Communication).
8 Osler, W.: "Principles and Practice of Medicine," edited by Thomas McCrae Appleton, New York, 1925, p. 1155.
9 Norris, G. W. and Landis, H. R. M.: "Diseases of the Chest and the Principles of Physical Diagnosis," W. B. Saunders Company, Philadelphia, 1918 Edition, p. 543.
10 Joannides, M. and Schlack, O. C.: "Use of Phrenic Neurectomy Combined with Artificial Pneumoperitoneum for Collapse of Adherent Tuberculous Lung," *J. Thoracic Surg.,* 6: 218, 1936.

11 Neffziger, H. C.: "The Scalenus Syndrome," *Surg., Gynecol. and Obst.*, 64: 119, 1937.
12 Joannides, M.: "The Necessity of Routine X-ray Studies of the Chest in General Hospitals," *Modern Hospital*, 53: 59, 1939.

Discussion

ALVIS E. GREER, M.D., F.C.C.P.
Houston, Texas

Dr. Joannides' paper warning us against "snapshot" diagnoses of painful conditions about the costoabdominal area is timely and valuable. Diagnostic problems of this kind have undoubtedly been encountered by all of us. I recall seeing a patient, many years ago, who developed ponopnea, fever, immobilization of the right lower hemithorax, spasm of transversus abdominalis muscle and leucocytosis, in whom a diagnosis of acute cholecystitis was made. No abnormal findings were noted at operation and the patient recovered rapidly thereafter. Since that time I have seen a number of cases of acute diaphragmitis. I have experienced difficulty in eliminating acute diaphragmatic pleurisy, although in my observations I have never seen decided flattening of the diaphragm in acute diaphragmatic pleurisy. The other symptoms of Hedblom's Syndrome may be present although the pain in acute diaphragmatic pleurisy has more of a spasmodic character. Dr. Joseph Capps, during the time I was interne in Cook County Hospital, first called attention to the difference in radiation of pain dependent upon whether the central or peripheral portions of the diaphragm were stimulated. In my own personal experience the most valuable differential factors in the diagnosis of acute diaphragmitis are immobility and flattening of the diaphragm and a tonic rigidity of the transversus abdominal muscle of the affected side. One would expect the left diaphragm to be involved as frequently as the right one, but all the cases I have observed have been on the right side. Why this is true I do not know. In the examination of roentgenograms of patients with chronic pulmonary disease, especially non-tuberculous types, we have repeatedly seen fixed, thickened, flattened diaphragmatic contours, and in many cases with only slight evidence of any active intrapulmonic disease.

Dr. Joannides has made a valuable contribution by his scientific study of these cases and he is to be commended highly for bringing this subject again to our attention.

The Unexpandable Lung*

SYDNEY JACOBS, M.D., F.C.C.P.**
New Orleans, Louisiana

Artificial pneumothorax is generally regarded as a reversible procedure, and usually it is. Comparatively little attention has been directed however, to the fact that when it is stopped, very seldom does the lung fully re-expand. Pinner, Leiner and Zavod[9] remark that the term "re-expansion" is a misnomer. Often the affected portion of the lung remains permanently collapsed, and the portion of the hemithorax thus vacated is filled variously, with fluid, by compensatory emphysema of the same or of the opposite lung, by elevation of the diaphragm, mediastinal shift or retraction of the chest wall. In all of these instances, the pleural space is obliterated, or else there is left a "dead space" between the two layers of the pleura, and the lung is said to be unexpandable.

This complication is recognized now more frequently than before, but we still do not know how often it occurs. Farber,[1] on the basis of an admittedly limited series, estimates that about 5 per cent of lungs successfully collapsed by pneumothorax cannot be made to re-expand. This paper does not represent a statistical study; it is a report of a few cases to emphasize the importance of the clinical problem and to point to the fact that artificial pneumothorax is not always a "temporary" treatment.

When a lung fails to re-expand,[2] generally it is because of any one or any combination of three factors: bronchial obstruction, massive pulmonary fibrosis and thickened visceral pleura.

Bronchial obstruction may be caused by external pressure but usually it means stenosis secondary to tuberculous endobronchitis.[7] Distal to the bronchostenosis, whatever the cause, stasis and accumulation of septic products and absorption of the entrapped air produce progressive atelectasis of the affected portion of the lung, and it is incapable of re-expanding.[4] Only too often empyema occurs as a complication of bronchial stenosis.

Massive pulmonary fibrosis may arise independently of bronchial obstruction by healing of large tuberculous areas. This

---

*Read before the Southern Chapter, American College of Chest Physicians, St. Louis, Missouri, November 13, 1944.

**From the Department of Medicine, School of Medicine, Tulane University of Louisiana and the Charity Hospital of Louisiana.

combination of infection plus disease can prevent re-flation of the lung. As Lindbloom pointed out, fibrosis affects the interlobular septa, gradually leading to organization of most or all of the involved lung. This process of carnification cannot be reversed; consequently, even after all the air in the pneumothorax space has been exhausted, the lung will not re-expand.

The most common hindrance[4] to re-expansion is the presence of a universally thickened visceral pleura with fibrous septa leading into the interior in all directions. Even in the absence of bronchial obstruction, such a lung cannot re-expand because the pleura, which has been enormously thickened by prolonged empyema (or occasionally hemothorax or hydrothorax), is too tough. Generally some degree of bronchial stenosis is associated with this, but even without it, air entering the bronchi under atmospheric pressure cannot distend a lung bound together by the tough fibers.

CASE I

L. C., a 23 year old white man, was first seen April 6, 1941, two days after a serious hemoptysis. Artificial pneumothorax was induced on the left side May 10, 1941. The lung was almost completely collapsed by November 11, 1941. Fluid was first noted 7 months after pneumothorax had been started. On two occasions 500 cc. were aspirated. He was last given a refill on December 10, 1943. Because of contralateral disease, an attempt was made to re-expand the left lung, but there is no obvious change in the roentgenological appearance after 10 months. His vital capacity is 1150 cc.

CASE II

E. G., a 36 year old white man was first seen January 16, 1941. Pneumothorax was started on the left side February 5, 1941, and almost complete collapse was noted on April 2, 1941. Fluid was first detected on June 14, 1941 and was removed repeatedly in 1943.

All refills of air were abandoned but no re-expansion of the lung is evident. Because the pleural space could not otherwise be obliterated, left phrenic crushing was performed on May 19, 1944, but the patient is still left with a "pleural dead space."

CASE III

E. H., a 19 year old white man was first seen February 15, 1939 for secondary syphilis for which he accepted only a few injections of an arsenical. He reported on April 26, 1940 because of hemoptysis; pneumothorax was started on the left side on May 11, 1940. Fluid was aspirated several times during 1941. Because of spread to the contralateral side, pneumothorax was started on the right side while attempts were made to re-expand the left lung in 1942. It was necessary to aspirate fluid repeatedly from the right side in 1943 and 1944. Attempt to re-expand both lungs have been unsuccessful, a pleural "dead space" existing bilaterally. His vital capacity is 1250 cc. (38 per cent of the computed normal).

CASE IV

L. W., a 27 year old white woman was first seen October 3, 1939 when she was 2 months pregnant. Because of a rapidly progressive lesion of the right lung, artificial pneumothorax was induced on December 5, 1939. Fluid was first noted on February 24, 1940. Repeated aspirations were necessary, complete collapse being evident November 8, 1940. By January 1942, there was complete re-expansions of the lower two-thirds and partial re-expansion of the upper third of the lung but a small amount of fluid persisted despite frequent attempts to remove it. The interval between treatments was extended to 10 weeks but at the end of this time, the patient complained of intense dyspnea and thoracic pain relieved only by a "refill". The lung has apparently not re-expanded at all in the past 30 months.

CASE V

N. L., a 43 year old white female, was first seen March 15, 1937 because of disease localized to the left upper lobe. After 6 months of conservative therapy, there was no change in the size of the cavity, and acid-fast bacilli were still present in the sputum; so artificial pneumothorax was induced. Adhesions preventing adequate collapse were severed 4 months later, and complete collapse of the entire lung ensued. Pure tuberculous empyema followed and persisted with febrile symptoms about 8 months, after which time, the patient became ambulatory. A small amount of fluid re-appeared from time to time, but no tubercle bacilli were isolated from it. Subsequently, 50 to 75 cc. of air administered every 15 to 20 days caused a marked rise in intrapleural pressure with moderate dyspnea. In 1940 attempts were made to re-expand the lung by prolonging the inter-treatment intervals to 12 weeks, but no re-expansion was evident roentgenologically. In 1941 treatments were suspended for a period of 7 months. At the end of that time, alarming symptoms of paroxysmal dyspnea and respiratory embarrassment began. The intrapleural pressure became exceedingly negative; it was necessary to administer oxygen by mask. Treatments were resumed and were given daily at first and subsequently at gradually lengthening intervals after which the respiratory embarrassment disappeared. In 1943 the suspicion of bronchopleural fistula was entertained but was not sustained by gas analysis. The pleural space was eventually obliterated by a complete thoracoplasty.

If one attempts to re-expand a lung by discontinuing "refills" and finds no evidence of resumption of the former volume, generally one of two things happens. Either the pleural space is obliterated by one of the factors previously mentioned (usually formation of fluid or deviation of the mediastinal structures) or alarming symptoms ensue after a variable period of time. The intrapleural pressure becomes increasingly negative leading to respiratory and circulatory embarrassment. The patient becomes intensely dyspneic, there is a marked tachycardia, and circulatory collapse rapidly develops. That these symptoms are due to absorption of gas from the pleural space and to traction on the mediastinal vessels is indicated by the fact that they rapidly abate as soon as the intrapleural pressures have been brought back almost

to atmospheric by instillation of air. In a sense, these symptoms are only exaggerated counterparts of those reported by many patients when the interval between refills is unduly extended. Any attempt at vigorous re-expansion of these unexpandable lungs is followed by dangerous consequences.

RECOGNITION OF THE UNEXPANDABLE LUNG

One may be warned of the imminence of the unexpandable lung by certain signs during the course of pneumothorax therapy. Small amounts of air given at a refill soon cause the post-treatment pressures to become positive; the patient needs less and less air at succeeding refills, and even with smaller amounts of air, it is possible to extend the time interval between treatments. It can also be noted at fluoroscopic examinations (and verified on roentgenograms) that there is comparatively little change in lung volume on maximal inspiration and forced expiration. After each refill, the patient complains of dyspnea.

Under these circumstances, one should at once make efforts to re-expand the lung before the irreversible phase is reached. All possible fluid should be aspirated. Bronchoscopy should be employed to determine if there is any degree of bronchostenosis which can be overcome at least long enough to permit some re-expansion of the lung.

What shall we do when a lung becomes unexpandable? The easiest way to manage this problem is simply to continue pneumothorax for the life-time of the individual. That this may not be the safest course to pursue is obvious. Pneumothorax is not efficient as a means of permanent collapse; there are always the dangers of rupture of the lung and recurrent empyema. Until a large series of patients receiving pneumothorax for a long period of time (10 to 15 years for example) has been watched to learn the indidence of these complications, we do not really know the extent of this danger. In all probability if the "dead space" is small, thoracoplasty is preferable; if the "dead space" appears too large to be collapsed easily by thoracoplasty, pneumothorax offers fewer hazards. It must be admitted that at the present time we have very little recorded experience to guide us.

PREVENTION OF THE UNEXPANDABLE LUNG

Possibly one can prevent this complication by better selection of cases and by more careful attention to details of the management of the pneumothorax patient. Inasmuch as bronchostenosis is one of the most common antecedents of the unexpandable lung, one should hesitate to perform artificial pneumothorax where bronchostenosis is impending.[7] This means that it should not be

performed if there is strong suspicion of tuberculous endobronchitis. Unilateral wheezing, otherwise unexplained dyspnea, large amounts of sputum with marked daily variations in volume or x-ray evidence of concentration of disease about the root of the lung[8] are symptoms, any of which should cause one to be extremely reluctant to induce pneumothorax. Unfortunately one cannot usually detect lesser degrees of bronchostenosis without bronchoscopy. It has been suggested that bronchoscopy[6] should always be performed before artificial pneumothorax is induced. This may be an ideal prerequisite, but it is not as yet feasible in most places.

To minimize thickening of the visceral pleura, attempts should be made to reduce the effect of pleural effusions. This, of course, implies frequent aspirations. Selective collapse, where attainable through the application of collapsing (relaxing) rather than compressing pressures, may conserve the function of uninvolved lung and tend to promote eventual re-expansion.

SUMMARY AND CONCLUSION

1. There are times when a lung fails to re-expand after being collapsed by artificial pneumothorax.

2. This unexpandable state may be due to bronchial obstruction, massive fibrosis of the lung, protracted empyema or to any combination of these.

3. Early detection of this complication is possible by frequent fluoroscopic examinations and manometric observations.

4. Perhaps this complication would be encountered less frequently if all pleural effusions of any magnitude were removed by frequent aspirations, if a collapsing (relaxing) compressive type of pneumothorax were maintained, and if artificial pneumothorax were not used where bronchial stenosis exists.

5. Treatment of the unexpandable lung calls for obliteration of the pleural space by thoracoplasty or by oleothorax. If neither one is feasible, refills must be indefinitely continued.

I am grateful to Drs. Maurice Campagna and Julius Wilson for permission to report cases from their service and to Mrs. Lorraine Birsner, medical social worker, for assistance in compiling data.

RESUMEN Y CONCLUSION

1. A veces no se puede obtener la reexpansión del pulmón después de haber sido colapsado por el neumotórax artificial.

2. Esta falta de expansión puede ser causada por obstrucción bronquial, fibrosis pulmonar masiva, empiema prolongado o cualquiera combinación de estos estados.

3. Mediante frecuentes exámenes radioscópicos y observaciones

manométricas es posible el descubrimiento temprano de esta complicación.

4. Tal vez sobrevendría esta complicación con menos frecuencia si se practicara la aspiración frecuente de todos los derrames pleurales de cierta magnitud, si se aplicara un neumotórax de tipo colapsante (dilatador) y no compresivo, y si no se empleara el neumotórax artificial en casos en los que existe estenosis bronquial.

5. El tratamiento del pulmón falto de expansión exige la obliteración del espacio pleural mediante la toracoplastia o el oleotórax. Si ni el uno ni el otro son factibles deben continuarse las insuflaciones indefinidamente.

REFERENCES

1 Farber, J. E. and Lincoln, N. T.: "Unexpandable Lung; Statement of Problem," *Am. Rev. Tuberc.*, 40: 704-709, (Dec.) 1939.
"Unexpandable Lung; Case Reports," *Am. Rev. Tuberc.*, 40: 710-721, (Dec.) 1939.
2 Farber, J. E.: "Unexpandable Lungs; Report of 27 Cases," *J. Thoracic Surg.*, 11: 424-433, (Apr.) 1942.
3 Farber, J. E.: "Bronchial Stenosis and Unexpandable Lungs; Report of 2 Cases," *Am. Rev. Tuberc.*, 43: 779-784, (June) 1941.
4 Eglee, E. P. and Wylie, R. H.: "Empyema and Unexpanded Lung Following Artificial Pneumothorax for Tuberculosis," *J. Thoracic Surg.*, 10: 603-617, (Aug.) 1941.
5 Potter, B. P.: "Study Concerning Clinical and Anatomic Features of Re-expanded Lungs Which had been Collapsed by Pneumothorax for Variable Periods of Time," *J. Thoracic Surg.*, 11: 554-564, (June) 1942.
6 Rafferty, T. N.: "Artificial Pneumothorax; Nonstatistical Analysis of Major Factors Involved in its Proper Management," *J. Thoracic Surg.*, 12: 578-589, (Aug.) 1943.
7 Rafferty, T. N. and Shields, D. O.: "Management of Pulmonary Tuberculosis Complicated by Bronchial Tuberculosis, With Special Reference to the Use of Artificial Pneumothorax," *J. Thoracic Surg.*, 12: 225-233, (Feb.) 1943.
8 Tuttle, W. M.; O'Brien, E. J.; Day, J. C. and Phillips, F. J.: "Tuberculous Stenosis of the Major Bronchi," *J. Thoracic Surg.*, 11: 299-307, (Feb.) 1942.
9 Pinner, M.; Leiner, G. and Zavod, W. A.: "Bronchospirography. The Functional Capacity of Normal Lungs, Severely Damaged Lungs, Lungs with Strictly Parenchymal Lesions, Thoracoplasty Lungs and Re-expanded Pneumothorax Lungs," *J. Thoracic Surg.*, 11: 241-265, (Feb.) 1942.

Artificial Pneumothorax and Its Effect Upon the Pulmonary Tissues of Normal Rabbits*

ROBERT CHARR, M.D., F.C.C.P.; HEINZ LORGE, M.D., F.C.C.P. *and* NATHAN H. HEILIGMAN, M.D., F.C.C.P.

White Haven, Pennsylvania

The purpose of this investigation was to note whether an artificial pneumothorax given over a considerable length of time would produce pulmonary fibrosis, vascular sclerosis, bronchial occlusion and pleural thickening.

Material Studied:

With this view in mind, pneumothorax was given to the left side of twelve normal rabbits. A small pneumothorax machine after the Robinson model was constructed. Short bevelled 19-gauge needles were used to introduce the air into the pleural space. Anesthesia was not used. The skin over the left side of the thorax was shaved and cleansed with soap and water; then iodine was applied which was later washed with alcohol. The needle was slowly inserted into the intercostal space. When the needle point entered the pleural space negative pressures 4 to 5 cm. in water were noted. When this happened, air was cautiously introduced. At the time of the initial injection, the amount given varied between 10 to 50 cc. of air. During the first two weeks of the experiment, the animals received daily injections. From the third week the refills of air were given every other day. Beginning from the second month, the injections were given every five days to a week. All the animals were examined daily by fluoroscopy to make sure that the collapse of the lung was satisfactory (Fig. 1). The duration of pneumothorax in this series varied from one week to ten months. In four animals the collapsed lungs were allowed to expand completely before death, with a view to determining whether the lungs once collapsed would return to normal appearance.

Gross and microscopic studies of both the collapsed and the non-collapsed lungs were made. The sections were stained with hematoxylin and eosin, Van Gieson, Weigert and Mallory stains.

---

*From the Pathological Laboratory of the White Haven Sanatorium, White Haven, Pennsylvania.

Findings:

The principal findings in this series was the absence of pulmonary fibrosis, vascular sclerosis and structural changes of the bronchi. Even in the animals which received the pneumothorax as long as 10 months, the collapsed lungs failed to show pulmonary fibrosis, vascular sclerosis or bronchial occlusion.

All the animals showed pleural changes on the side of the pneumothorax. The normal glistening lustre of the pleura was replaced by dull, fibrinous covering. Scraping the matter off the surface revealed an underlying inflamed pleura. In two animals which had the pneumothorax for ten months, the thickness of the pleura equalled that of the cross section of the upper lobes of the collapsed lungs (Fig. 2).

In all the animals the collapsed lung was dark red, much de-

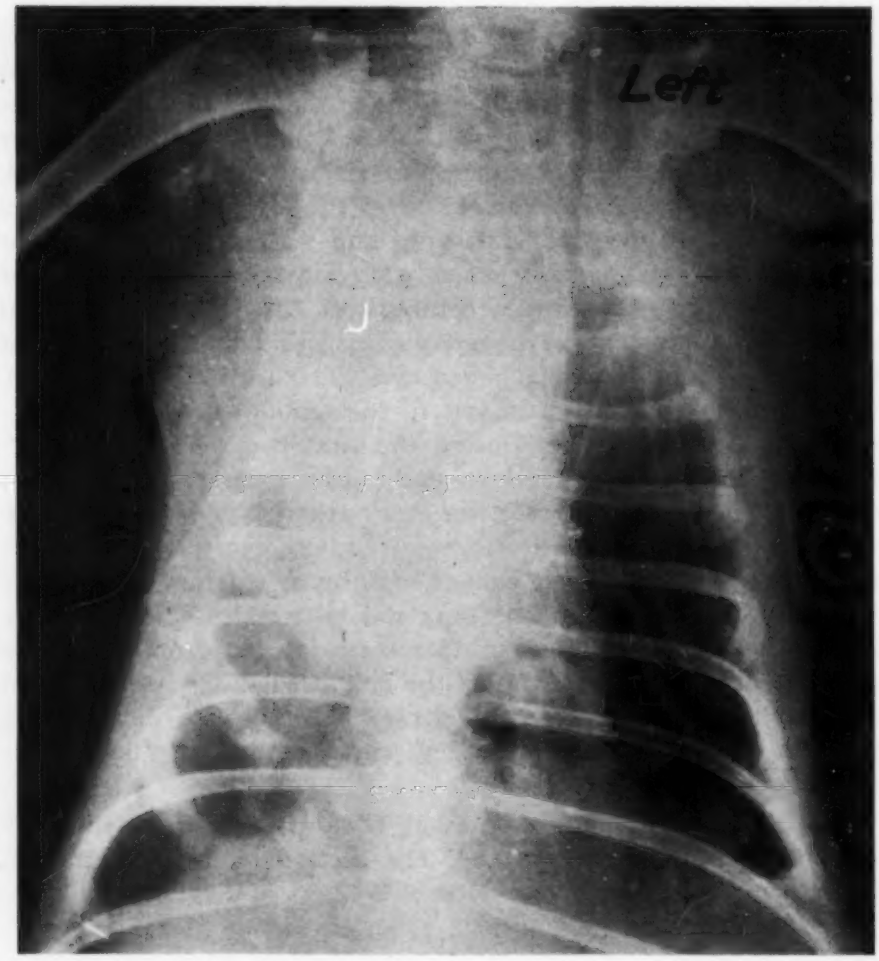

Figure 1: A roentgenogram of a rabbit with pneumothorax on the left side.

creased in size and fleshy in consistency. The collapse of the alveoli was most marked in the central portions of the lung rather than in the periphery (Fig. 3). The blood vessels in the center of the collapsed lungs were engorged whereas those in the periphery were relatively empty. In the lungs which had been allowed to re-expand, the re-expansion was more complete and rapid in the peripheral zones of the lung. Even in those animals which roentgenologically showed no pneumothorax whatsoever remaining upon autopsy, there was found considerable collapse and vascular engorgement in the central portions of the lungs.

The non-collapsed right lungs showed the following changes: In the animals which were killed at the end of one month of pneumothorax, the non-collapsed lungs showed extreme engorgement with blood. Not infrequently subpleural and alveolar hemorrhages were noted. There was no evidence of emphysema. In the animals which received pneumothorax for six months, the non-collapsed lung still showed vascular engorgement but to a less degree. Still there was no marked emphysematous change. In the

Figure 2: A cross section of the left lung of a rabbit with 10 month old pneumothorax showing thickening of the pleura.

animals which received pneumothorax for ten months the non-collapsed lungs were emphysematous and relatively avascular.

SUMMARY

In the series studied, the pneumothorax caused pleural thickening but no pulmonary fibrosis, vascular sclerosis or bronchial occlusion in ten months.

The alveolar collapse was most marked in the central portions of the lungs, where the blood vessels were engorged. Re-expansion was faster in the periphery of the lungs.

The non-collapsed lungs gradually became emphysematous.

RESUMEN

En la serie estudiada el neumotórax causó engrosamiento pleural, pero no ocasionó fibrosis pulmonar, esclerosis vascular u oclusión bronquial durante diez meses.

El colapso alveolar fue más marcado en la parte central del pulmón, donde se encontraban congestionados los vasos sanguíneos. La reexpansión fue más rápida en las zonas pulmonares periféricas.

Los pulmones no colapsados gradualmente se volvieron enfisematosos.

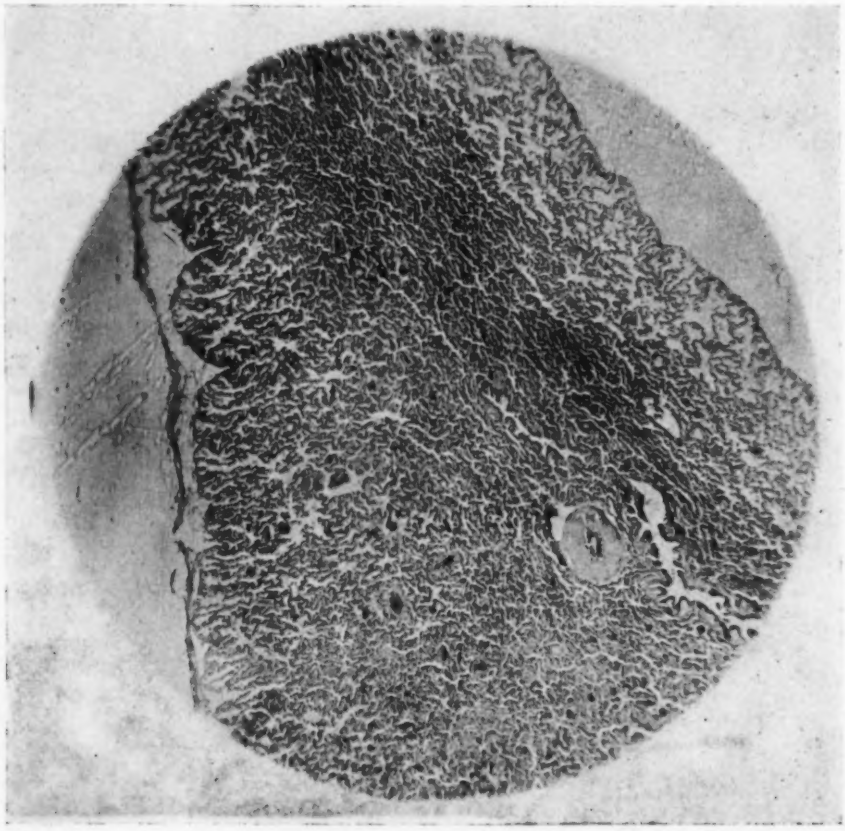

Figure 3: A cross section of a collapsed lung showing more collapse and vascularity in the center of the lung.

Induced Pneumoperitoneum in the Treatment of Advanced Pulmonary Tuberculosis in Children*

EVERETT MORRIS, M.D., F.C.C.P.**
Olive View, California

The purpose of this study is to determine the effectiveness of induced pneumoperitoneum in the treatment of children suffering from advanced pulmonary tuberculosis and with poor prognoses.

Among 40 pneumoperitoneum cases treated here there had been done in advance, but unsuccessfully, 37 intrapleural pneumothoraces of which 6 were bilateral, 30 phrenic interruptions including 8 that were repeated and 3 evulsions. In 2 desperately ill children no other method of collapse therapy was thought advisable. This study dates back to December, 1936. The youngest patient was ten years of age and the oldest less than eighteen years.

TABLE I

Therapeutic Pneumoperitoneum

Total number of cases		42
Males	25	
Females	17	
Average age 14½ years		
Of Spanish descent		76.6%
Negroes		14.2%
Whites		4.7%
Japanese		2.4%

TABLE II

Classification—N.T.A.

Mod. Adv. 15 — Far Adv. 27	
Indications — Other compression methods ineffective. Average length treatment in months	9.1
Average No. treatments	41.8
Average No. C.C. of Air per treatment	725
Average Manometric Readings	+5.8: +8.4 Cm's.

TABLE III

Post Operative Symptoms and Operations

Dyspnea	3	Anorexia	6
Pain	14	Loss of weight	19
P.O. Phrenic Crush			12
P.O. Extrapleural Pneumo.			4
P.O. Thoracoplasty			3
Concurrent Bilat. Intrapl. Pnx.			1
Concurrent Unilat. Intrapl. Pnx.			1

The post operative temporary phrenic cases are classified as follows: 3 have become arrested; 3 improved; 3 unimproved and 3 have died. Only 3 of these cases were not operated prior to the

*Read before the Trudeau Society, Los Angeles, California, May 16, 1944.
**Olive View Sanatorium, Olive View, California.

induction of pneumoperitoneum. One was unimproved and two, now hospitalized, are quiescent.

Of the four extrapleural pneumothoraces, one experienced an appendectomy after four months treatment. The appendix, however, was not tuberculous. After an interval of two months pneumoperitoneum was re-established and continued for three months, when a roentgenogram showed two cavities visible in the right lung which were not present previously. The diaphragm on this side had risen 6½ cm. and on the left 5 cm. The pneumoperitoneum was definitely ineffective. A right extrapleural pneumothorax was established and refills were continued for ten months when the patient was discharged as quiescent. The collapse was maintained at a clinic for four years. The patient has married, has a baby and is doing her housework.

The second child was a Japanese girl 14 years old when admitted. Previously, there had been an unsuccessful attempt to establish a left intrapleural pneumothorax and a left phrenic interruption. A poor pneumoperitoneum was established. On admission the pneumoperitoneum was discontinued as ineffective and a left intrapleural pneumothorax was recommended which was mechanically unsuccessful. Nine months later an extrapleural pneumothorax was established on the left and refills were continued, without incident, for three months when an appendectomy was done. The appendix was tuberculous, also there was a generalized tuberculous peritonitis. This was sixteen months after pneumoperitoneum had been discontinued. It was re-established three weeks later and continued uninterruptedly for three months when it was discontinued again as ineffective. The extrapleural pneumothorax was continued for 39 months after pneumoperitoneum had been discontinued. Hospitalization has been continued in an outside sanatorium. Her prognosis is grave.

In this 12 year old boy a right phrenic crush had to be repeated, also a right intrapleural pneumothorax was mechanically unsatisfactory. Pneumoperitoneum was established at Olive View and 155 refills were given. During this period two cavities closed but a small one remained open. It was decided to discontinue the treatments as ineffective. After a period of six months bed rest an extrapleural pneumothorax was established and it has been continued for two years. His sputum remains positive and his prognosis is guarded.

One of the post operative thoracoplasty cases is a 16-year-old white boy who received pneumoperitoneum for 34 months. Three cavities in the right mid lung field, measuring 2½ cm., 3 cm., and 3 cm., and one measuring 1 cm. in the left mid lung field were closed after 2½ years of treatments. While insufflations were

being continued a cavity reappeared. The cavity was present three months later when an extrapleural pneumothorax was substituted and maintained for ten months. Following this a first, second and third stage thoracoplasty was done. His sputum is positive and his prognosis is grave.

Another post operative thoracoplasty was done on a boy who on a previous admission had been treated successfully with pneumoperitoneum for 21 months. When discharged two cavities in the upper third of each lung had been closed and the sputum had been converted. He failed to report at the clinic for refills as recommended by the staff. He also failed to carry out the other post sanatorium instructions and was readmitted eight months later with a lesion requiring a first stage thoracoplasty. His sputum has been converted, but his prognosis is guarded.

The third post operative thoracoplasty case was done as the result of an unsuccessful pneumoperitoneum of six months duration. While no credit can be given to pneumoperitoneum, it is gratifying to know this patient has been economically independent for six years.

In another case an established bilateral intrapleural pneumothorax was continued for 13 months after a concurrent pneumoperitoneum of three months duration was discontinued as ineffective. Prior to the establishment of the pneumoperitoneum the following had been done: 3 phrenic crushes, an evulsion and a bilateral intrapleural pneumothorax. Her sputum was converted. She died after 5½ years of hospitalization.

In one case pneumoperitoneum was advised in order to supplement a right intrapleural pneumothorax of 11 months duration, complicated by a pleural effusion. He was discharged after 23 months as improved and with a good prognosis. Subsequently the pneumothorax was discontinued at the clinic after 27 months and the pneumoperitoneum after 15 months. These collapse therapies were discontinued 55 and 28 months respectively before this young man entered the U. S. Army Air Forces, where after three months of training an active lesion was discovered in his left lung. He is now in a Veterans Administration Hospital.

TABLE IV
Sputum Studies

Pre-operative			
Positive	38	Negative	4
Post-operative		Conversions	6
Positive	26	Negative	5
		Sputum free	4
		Unknown	1

SPUTUM

Of the four pre-operative negative sputum cases reported three subsequently became positive and one remained negative. In this case eight futile attempts were made to secure satisfactory sputum specimens. This was before stomach washing for the demonstration of tubercle bacilli had been adopted as a routine method in such cases. However, a diagnosis of pulmonary tuberculosis was established by our roentgenologist who found two small cavities in the left apex. These closed after the 39th pneumoperitoneum refill and remained closed. The treatments were continued for a total of 21 months. The patient is now classified as arrested.

In addition to the six conversions one patient was reported as having three negative concentrations and cultures. In two cases there were reported five or more negative 24-hour concentrates, one with three and one with two negative concentrates; four became sputum free; this was before stomach washing was a routine procedure. The sputum remained positive in 14 cases. Among those who died two were converted, 11 remained positive and in one case there is no post operative sputum report.

After the demonstration of tubercle bacilli in the sputum, two 24-hour concentration sputum specimens are studied 7 days apart. If both are negative another concentration and a gastric lavage specimen are taken simultaneously. If these are culturally negative the sputum is said to be converted.

TABLE V
Complications

Pneumatocele	1
Temporary spasms of diaphragm	1
Tuberculous Peritonitis	4
Tuberculous Enteritis	4
Ischio-rectal fistula	1

COMPLICATIONS

There were no fatal or alarming accidents during or following the administration of pneumoperitoneum. It appears that peritoneal effusions are not as frequent complications, even in small

TABLE VI
14 Deaths

Before PN'P.			*After PN'P.*			
Temporary Phrenic Block	*Permanent Phrenic Block*	*Intra-Pleural Pneumothorax*	*Temporary Phrenic Block*	*Intra-Pleural Pneumothorax*	*Extra-Pleural Pneumothorax*	*Thoraco-plasty*
11	2	11	15	4	4	1

amounts, as pleural effusions complicating induced pneumothorax. Intraperitoneal adhesions are not uncommon.

On admission the 14 children who died were classified by our roentgenologist as far advanced with cavitation. Ten died in the institution. Either before or after admission to Olive View there had been attempted or completed 11 temporary phrenic nerve interruptions including a bilateral case and two permanent phrenics. Likewise, there were done 11 intrapleural pneumothoraces, five of which were bilateral. After the induction of pneumoperitoneum 15 temporary phrenic nerve crushes were done. Intrapleural pneumothoraces were done in four, extrapleural pneumothoraces in 4 and a thoracoplasty in one. In three cases pneumoperitoneum proved to be clinically harmful.

AUTOPSIES

Four of these cases came to autopsy. Pneumoperitoneum was discontinued a fortnight before the death of one child and the autopsy revealed "About 200 cc. of clear fluid in the peritoneum, and the free surfaces of the peritoneum appear thickened or cloudy over the spleen, liver, mesentery, etc. There was a small yellow nodule in the peritoneum just over the liver region about 1 mm. in diameter."

In the second patient pneumoperitoneum was given six days before the demise. At autopsy "A small amount of free air was found in the peritoneum." No mention was made of any peritoneal involvement.

In the third case of this series, pneumoperitoneum was discontinued as ineffective after 10 refills. An autopsy was done 35 months later and the report reads: "All peritoneal surfaces are smooth and glossy."

In the fourth case pneumoperitoneum was discontinued as unsatisfactory after the 65th refill. Six months later the patient died and at autopsy the following was found: "The entire peritoneal surfaces appear dry and thickened but show no gross tuberculosis or other lesions."

In the group of 10 pneumoperitoneum cases classified as effec-

TABLE VII
10 Effective PN'P.

Before PN'P.		*After PN'P.*	
Temporary Nerve Block	10	Temporary Nerve Block Repeated	4
Intra Pleural PN'P.	9	Initial Temporary Nerve Block	2

tive, all showed unsatisfactory results from traumatization of the phrenic nerves before pneumoperitoneum was instituted, and in 4, crushes were repeated after pneumoperitoneum had been started with indifferent results. In 2, initial crushes were done following the insulflation with good results. In this same group 9 unsuccessful intra-pleural pneumothoraces were done before and none after the initial pneumoperitoneum. The average number of months of hospitalization for the 8 discharged patients was 34, and the average duration of the pneumoperitoneum was 24 months. For the two boys now hospitalized the averages are 3 years and 2 years respectively.

ROENTGEN STUDY

The stereoroentgenograms taken of the 42 children reveal unilateral cavitation in 25, bilateral cavitation in 9 and in 8 no cavitation was visualized. There was a total of 69 cavities ranging in size from ½ cm. to 6 cm.

In the group of 10 effective pneumoperitoneum cases 5 showed cavities located in the upper third of the right lung; 1 had bilateral cavitation in the same region; 1 had 4 cavities in the right middle and lower third of the lung; 2 had 6 cavities in the left upper third and 1 revealed a cavity in the right lower third of the lung. The diaphragms were elevated from 2 cm. to 10 cm.

CLINICAL AND ECONOMIC RESULTS

A summary of the clinical and economic results in the 10 children whose disease was brought under control follows: Two were dis-

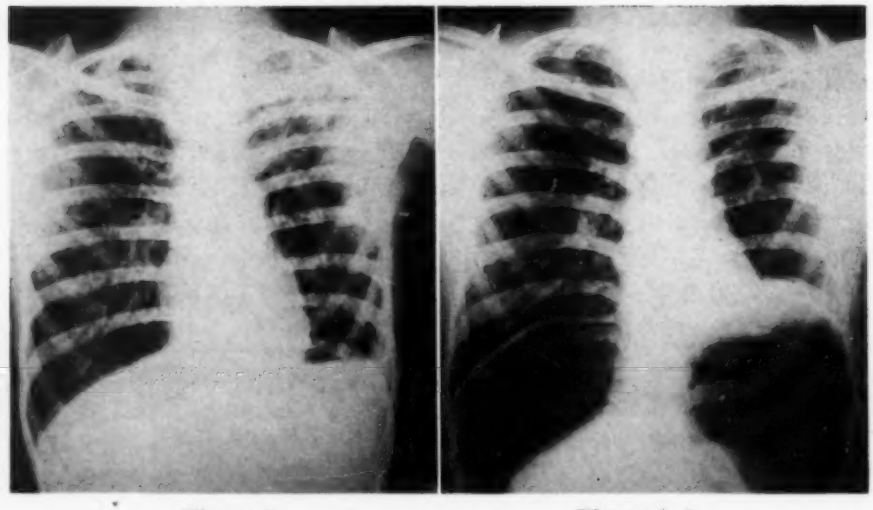

Figure 1 Figure 1-A

Fig. 1. Appearance of chest on admission. Compare with Fig. 1-A.—*Fig. 1-A:* Taken after the 86th refill. After the 2nd refill a left temporary phrenic crush was done. This boy is hospitalized and his disease is quiescent.

charged as improved, due to induced pneumoperitoneum, with the recommendations that they continue the treatments at a clinic. One followed instructions and is doing well. The other disregarded them and has been readmitted. There are two boys, hospitalized, whose lesions are quiescent but who continue to receive pneumoperitoneum. In each case a left temporary phrenic crush was done after collapse therapy had been instituted. In one a 2 cm. cavity and a questionable cavity were closed. They have remained closed 15 months. In the other, 4 cavities were closed and have remained closed for over one year. Sputum conversion has been accomplished in both cases. This type of pulmonary collapse therapy played an equal role with intrapleural pneumothorax and phrenic nerve block in 2 cases in which the disease was arrested. One was the air corps cadet and the other is a school girl.

This mode of treatment, we think, is responsible for the arrestment of the disease four years ago in a 14 year old school boy.

There are three arrested cases working in our war plants. The physician who treated one of these boys wrote on his chart, "Pneumoperitoneum certainly seems to be the answer to this boy's problem." The average length of time pneumoperitoneum was given these three boys was 33 months and the average length of time since discharge is 63 months. A careful analysis of their clinical progress here and the reports from the clinics lead us to believe that their recovery may safely be ascribed directly to the induction of pneumoperitoneum.

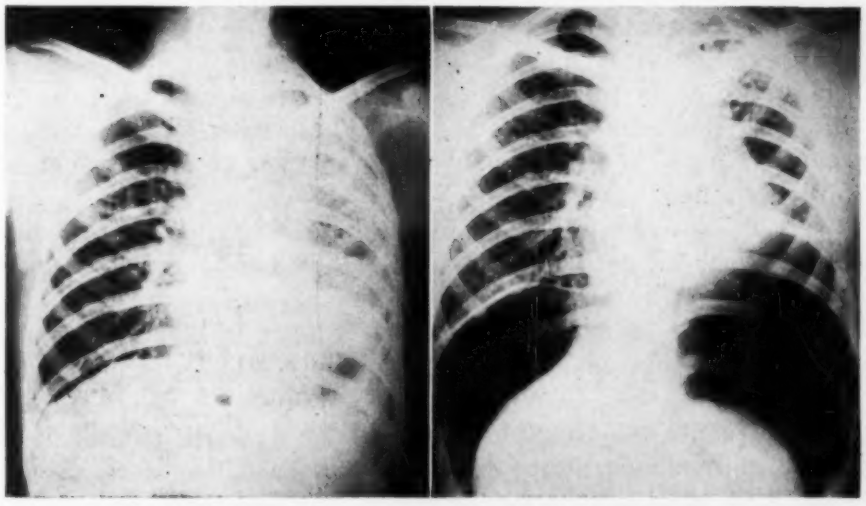

Figure 2 Figure 2-A

Fig. 2: Appearance of chest on admission. Compare with Fig. 2-A.—*Fig. 2-A:* Taken after the 98th refill. After the 9th refill a left temporary phrenic crush was done. The lesion is classified as quiescent. The boy is hospitalized.

CONCLUSIONS

This series of 42 patients, while not large, seems sufficient for illustration and the following tentative conclusions:

1. Artificial pneumoperitoneum is a mechanical measure that can be safely utilized independently or jointly with some of the other accepted methods of collapse therapy in the treatment of pulmonary tuberculosis in children.

2. In this limited number of cases analyzed, the location of the cavity or cavities had little to do with the effectiveness of pneumoperitoneum.

3. Pneumoperitoneum should be continued in successful cases perhaps for a longer period of time than intrapleural pneumothorax, as the air seems to be absorbed more rapidly in the peritoneal cavity than in the pleural sac. This is especially noticeable when patients are put on physical exercise.

4. The present clinical and economic status of the 10 patients in whom pneumoperitoneum was effective, warrants the conclusion that this measure has a definite but limited place, in carefully selected cases, and that it is effective in the treatment of children suffering from advanced pulmonary tuberculosis with poor prognoses.

CONCLUSIONES

Aunque esta serie de 42 enfermos no es grande, parece ser suficiente como ejemplo y para sacar las siguientes conclusiones provisionales.

1. El neumoperitoneo artificial es una medida mecánica que puede emplearse sin peligro, independientemente o combinada con algunos de los otros métodos de colapsoterapia aceptados, en el tratamiento de la tuberculosis pulmonar en los niños.

2. En el limitado número de casos analizados, la ubicación de la caverna o de las cavernas tuvo poco que ver con la eficacia del neumoperitoneo.

3. En los casos en los que es eficaz, debe continuarse el neumoperitoneo por un período de tiempo quizás más largo que el neumotórax intrapleural, pues parece que el aire se absorbe más rápidamente de la cavidad peritoneal que de la pleural. Se nota ésto especialmente cuando ya se les permite a los enfermos hacer ejercicio físico.

4. El presente estado clínico y económico de los 10 enfermos en los que el neumoperitoneo resultó eficaz justifica la conclusión de que este método tiene un lugar bien definido, aunque limitado, en casos cuidadosamente seleccionados, y de que es eficaz en el tratamiento de niños que sufren de tuberculosis pulmonar avanzada de mal pronóstico.

Laryngeal Tuberculosis*

M. K. HUMPHRIES, JR., M.D.**
Charlottesville, Virginia

In any tuberculosis institution the treatment of laryngeal tuberculosis occupies a prominent role in the therapy of this dreaded disease. The relationship of laryngeal tuberculosis to pulmonary tuberculosis is now better understood, and this knowledge has served to lessen the incidence of laryngeal complications and to assist in their eradication after they have appeared. The surgical treatment, as directed to the larynx, has likewise made progress, and beneficial results are now more uniformly obtained.

Drs. Hedges and Woodward made a study of such cases† in the five year period 1923-1928 and also for the five year period 1933-1938, and presented a comparative analysis. It occurred to me that it would be interesting to make a third five year study of laryngeal tuberculosis in the same institution for the period January 1st, 1939, through March 31st, 1944.

The study was undertaken to illustrate by comparative analysis the progress that has been made in the treatment of tuberculosis of the chest and of the larynx. There is a better understanding today of the disease, and great strides have been made in surgical therapy. Collapse therapy has been more widely used in this five year group, with good results both in the chest and laryngeal disease. The percentage of tuberculosis of the larynx has decreased greatly during the last five years. The likely reason for this is the increased rate of early diagnosis and the early institution of thoracic surgery.

There is some disagreement as to the manner of infection of the larynx; however, the consensus of opinion favors direct invasion by tubercle bacilli from sputum. Most patients with laryngeal tuberculosis do have a positive sputum, and the sputum in its passage from the lungs through the larynx keeps the posterior commissure constantly bathed with bacilli. The few cases of laryngeal involvement in which there is a negative sputum lend support

---

*Read before the Albemarle County Medical Society, Charlottesville, Virginia, October 5th, 1944.

**From the Dept. of Otolaryngology, University of Virginia Hospital.

†These Patients were under the care of Dr. Frank Stafford at the Blue Ridge Sanatorium, a Virginia State Tuberculosis Sanatorium. The laryngeal procedures were carried out by Dr. Glassell Slaughter FitzHugh and the writer, and the thoracic surgery by Dr. E. C. Drash and Dr. M. L. White of the University of Virginia Hospital.

to the theory of infection by the blood stream. There is general agreement that the sooner a negative sputum can be obtained, the sooner the laryngeal disease will improve.

The diagnosis of laryngeal tuberculosis in the early phase rests upon repeated mirror examinations of the larynx. The disease is often insidious in its development, and therefore periodic examinations are essential. The voice does not become hoarse except when the cords are involved or if their approximation is interfered with by a pathological condition in the posterior commissure. There may be pain or soreness if an ulcer is present, and this is aggravated by fruit juices and swallowing. In the far advanced cases occasionally swallowing is impossible without an anesthetic spray for relief of pain. This is the group which is helped most by surgical treatment. The general pathology is one of infiltration, edema, erosion, and ulceration. The more advanced the pathology, the more likely the process is to be extrinsic, i.e., involvement of the epiglottis, aryepiglottic folds, or the arytenoids. Intrinsic lesions (posterior commissure, vocal cords, ventricular bands, and false cords) require less cauterization and are not as painful.

The five year study, 1939-1944, revealed 139 cases of laryngeal tuberculosis in 2213 patients discharged from the Sanatorium, an incidence of 6.2 per cent. The table on comparative analysis will show that this figure is more than 50 per cent lower than in the previous studies. It is also in inverse ratio to the percentage of collapse therapy.

TABLE I

Age Incidence of Tuberculosis of the Larynx

	Male	*Female*	*Total*
Under 20	3	9	12
20 - 29	11	17	28
30 - 39	20	21	41
40 - 49	17	7	24
50 - 59	22	4	26
60 - 69	5	2	7
			139

The incidence among females is greater up to the age of 40; thereafter the incidence among males predominates.

The group ranged in age from the teens into the seventies; however, the majority of cases were in the twenty to sixty year old group. The ratio between males and females was slightly in favor of the males. The percentage was 56.5.

The study was carried further by dividing the 139 cases into three groups, depending upon the local pathology. Group I consisted of sixty cases of incipient tuberculosis of the larynx (Table II). In these cases there was no edema or ulceration. Most of the

TABLE II — GROUP I

Age Incidence of Tuberculosis of the Larynx
(Cases with no Edema or Ulceration)

	Male	*Female*	*Total*
Under 20	3	5	8
20 - 29	6	8	14
30 - 39	7	6	13
40 - 49	7	4	11
50 - 59	8	1	9
60 - 69	2	3	5
70 - 79	0	0	0
			60

Treatment of incipient cases: 1. Spray
2. Silence

Results of treatment of larynx and chest:

Larynx:	healed	41	*Chest:*	arrested	26
	improved	6		improved	20
	unimproved	13		unimproved	5
				dead	9

The incidence among females is slightly greater up to the age of 30; thereafter the incidence among males predominates.

patients complained of slight to moderate hoarseness, but 38.2 per cent were asymptomatic. A few had slight pain, soreness, and a feeling of fullness, due to the irritation from infiltration. Fifty-five per cent of this group were males. The age incidence ranged from the teens to seventy years, but the majority of patients fell in the twenty to forty year bracket. The pathology in this group involved only the posterior commissure in 80 per cent of the cases. It consisted of hyperemia and thickening of the mucosa, and some cases presented greyish elevations. In cases with normal cords, hoarseness could be attributed to the faulty function of the arytenoids as a result of the posterior commissure infiltration. Examination of the chest revealed 8.4 per cent in the minimal group, 45 per cent moderately advanced and 46.6 per cent far advanced. Seventy-five per cent had positive sputum on admission and 11.6 per cent positive sputum on discharge. It is an interesting fact that 71.6 per cent had some form of chest surgery. The main complication (Table XVI), was enteritis, it occurred in

TABLE III

Location of Pathology of Larynx, Group I

1. Posterior commissure	48
2. " " , right arytenoid	1
3. " " , right true cord	1
4. " " , left true cord	1
5. " " , both true cords	1
6. " " , both true cords, rt. arytenoid	1
7. Epiglottis and pharynx	1
8. Both true cords	1
9. Right true cord, left false cord	1
10. Both true and both false cords	1
11. Left true cord	1
12. Anterior commissure and left true cord	1
13. Generalized	1
	60

The posterior commissure was involved alone in 80 per cent of the cases in Group I.

6.6 per cent of all cases in the group. Treatment consisted of comforting sprays and silence. The results showed 78.3 per cent of the larynges healed or improved and 76.6 per cent of the chests healed or improved.

The second group of patients includes 52 cases with slight edema and slight ulceration of the larynx. The main symptoms were moderate cough, hoarseness, pain, soreness, and a feeling of congestion. Only 5.7 per cent were asymptomatic. Males and females were of equal number (Table IV). The largest proportion of patients fell in the twenty to forty year old group. The pathology in this more advanced group involved the posterior commissure per se in only 19.2 per cent of the patients. The tendency here was for the pathological process to be both intrinsic and extrinsic. Examination of the chest revealed 98 per cent were in the moderately and far advanced group. Eighty-eight per cent had positive sputum upon admission and 21 per cent had positive sputum upon discharge. Of the entire group 96 per cent had some form of chest surgery and the remaining 4 per cent had bronchoscopies. The chief complication, again, was enteritis, which occurred in 11.5 per cent of these cases. The treatment of this group, in addition to silence and spray, consisted of galvanocauterization and nerve block. The two cases in which it was necessary to inject alcohol into the superior laryngeal nerve were more or less mor-

TABLE IV — GROUP II

Age Incidence of Mild Degree of Tuberculosis of Larynx
(Cases with Slight Edema and Slight Ulceration)

	Male	*Female*	*Total*
Under 20	1	2	3
20 - 29	4	6	10
30 - 39	8	13	21
40 - 49	4	3	7
50 - 59	6	2	8
60 - 69	2	0	2
70 - 79	1	0	1
			52

Treatment:
1. Silence 52
2. Spray 40
3. Galvanocautery 14
4. Nerve Block 2
5. Tracheotomy 0

Results of Treatment of Larynx and Chest:

Larynx:	healed	24	*Chest:*	arrested	7
	improved	14		improved	26
	unimproved	14		unimproved	5
				dead	14

The incidence among females predominates up to the age of 40; thereafter the incidence of males is greatest.

TABLE V

Location of Pathology of Larynx, Group II

1. Posterior commissure		10
2. Posterior commissure and left arytenoid	(one or both)	1
3. " " " right arytenoid	(one or both)	1
4. " " " both arytenoids	(one or both)	3
5. " " " left arytenoid and left true cord		1
6. " " " right arytenoid and right true cord		1
7. " " " both true cords		10
8. " " " right true cord	(one true cord)	3
9. " " " left true cord	(one true cord)	1
10. " " " right false cord, right true cord		1
11. " " " arytenoids and true cords		2
12. " " " both true and false cords		1
13. " " " both arytenoids, both false cords		3

(Continued on next page)

14.	"	"	"	epiglottis	1
15.	"	"	"	pharynx	1
16.	"	"	"	arytenoids, right aryepiglottic fold	1
17.	"	"	"	left arytenoid, left false cord	1
18.	"	"	"	right true cord, aryepiglottic folds	1
19. Both true cords					1
20. Left cord and right arytenoid					1
21. Epiglottis and arytenoids					1
22. Right false cord					1
23. Entire larynx					1
24. Epiglottis					1
25. Epiglottis and linguo-epiglottic fold					2
26. Anterior commissure					1
					52

The pathology in this group involved the posterior commissure only in 19.2 per cent of the patients.

ibund and died several weeks afterwards; however, they obtained immediate relief of pain. They had extensive extrinsic and intrinsic lesions, and in spite of cauterization were unable to eat because of severe referred pain. Cauterization was used to the larynx in 26.9 per cent of the cases. The results of treatment to the larynx and chest reveal the larynx either improved or healed in 73 per cent of the cases, and the chest condition either improved or arrested in 55 per cent of the cases.

The third group includes 27 patients with advanced edema and ulceration of the larynx. The symptomatology consisted of marked cough, hoarseness, dysphagia, aphagia, referred pain, and aphonia. None were asymptomatic. Seventy and three tenths per cent were males. The largest proportion was in the 30 to 60 year old group. The pathological processes present in the larynx can be seen by the table to have been extrinsic or mixed in 90 per cent of the cases (Table VII). Examination of the chest revealed the lesion there to be moderately advanced in 18.4 per cent and far advanced in 81.6 per cent. The sputum was positive on admission in 92.5 per cent and positive on discharge in 14.8 per cent (Table VII). The chief complications were, first, renal tuberculosis, and second, enteritis and rectal fistula. Of this group 55.5 per cent received some form of chest surgery. The treatment of the larynx consisted of silence, comforting sprays, cauterization (in 66.6 per cent) and tracheotomy (1 case). The larynx was healed or improved in only 33.3 per cent of the cases, and the chest condition was arrested or improved in

TABLE VI — GROUP III

Age Incidence of Tuberculosis of the Larynx
(Cases of Advanced Edema and Ulceration)

	Male	*Female*	*Total*
Under 20	0	1	1
20 - 29	1	3	4
30 - 39	7	1	8
40 - 49	5	0	5
50 - 59	7	2	9
60 - 69	0	0	0
70 - 79	0	0	0
			27

Treatment:
1. Silence 27
2. Spray 27
3. Galvanocautery 18
4. Nerve Block 0
5. Tracheotomy 1

Results of Treatment of Larynx and Chest:

Larynx:			*Chest:*		
	healed	4		arrested	4
	improved	5		improved	3
	unimproved	7		unimproved	3
	worse	11		dead	17

The incidence among females is greater up to the age of 30; thereafter the incidence among males predominates.

TABLE VII

Location of Pathology of Larynx, Group III

1. Posterior commissure and arytenoids	1
2. " " , arytenoids, epiglottis	3
3. " " , arytenoids, false cords	3
4. " " , left arytenoid, left false cord	1
5. " " , arytenoids, false cords, epiglottis	1
6. Entire larynx	3
7. Posterior commissure, both true cords	1
8. " " , right true and right false cord	1
9. " " , right false-right true cord, arytenoid aryepiglottic and linguo-epiglottic folds	1
10. Posterior commissure, left arytenoid, both true cords	1
11. Epiglottis	1
12. Right arytenoid and right false cord	1
13. Epiglottis, arytenoids, aryepiglottic and linguo-epiglottic folds	1

(Continued on next page)

14. Epiglottis, posterior commissure, false cords, and aryepiglottic and linguo-epiglottic folds	1
15. Epiglottis, left arytenoid, left aryepiglottic fold	2
16. Epiglottis, posterior commissure, both arytenoids	1
17. Pharynx and entire larynx	1
18. Pharynx, epiglottis, left arytenoid, left linguo-epiglottic fold	1
19. Pharynx, tonsils, posterior commissure, arytenoids, left false cord	1
20. Pharynx, soft palate, epiglottis, arytenoids, aryepiglottic fold and linguo-epiglottic fold	1
	27

The lesions of the larynx in those patients with advanced edema and ulceration is extrinsic or mixed in 90 per cent of the cases.

TABLE VIII

Location of Cauterization in Each Group and the Number of Times Applied

	I	*II*	*III*	*Total*
Posterior Commissure	1	8	5	14
Anterior Commissure		1	0	1
Epiglottis		11	23	34
Both Arytenoids		9	7	16
Left Arytenoid		1	10	11
Right Arytenoid		0	6	6
Pharyngeal Ulcer		1		1
Left False Cord			7	7
Aryepiglottic Fold, Right			5	5
Aryepiglottic Fold, Left			5	5
				96

One case in Group II had Formalin applied to larynx twice weekly for 8 months, beginning with ½ per cent strength.
Average age of death in the entire group was 40.8 years.
Twenty-three per cent of the 139 cases received electro-cauterization.

TABLE IX

Chest Lesions in Group	*I*	*II*	*III*
Minimal	5	1	0
Moderately advanced	27	11	5
Far advanced	28	40	22

Ninety-seven and seven tenths per cent of the entire group studied had moderately or far advanced chest lesions.

25.9 per cent of the patients. The reason for such low percentages is obvious when one considers the fact that 81.4 per cent of the 27 cases had far advanced chest lesions and that 92.5 per cent had positive sputums upon admission to the Sanatorium.

Table XVII illustrates the results obtained in the three five year groups. A comparative analysis is presented. The marked drop in the percentage of laryngeal tuberculosis has been enlarged upon; the early diagnosis and increased use of thoracic surgery to obtain negative sputums no doubt play the major role in the diminution of the incidence. Not many years ago Dworetzky reported the incidence as high as 25 per cent in a mixed study. The results in this group show a gain of 5.6 per cent in those

TABLE X

Sputum Examination on Admission

	Positive	*Negative*
Group I	45	15
Group II	46	6
Group III	25	2

TABLE XI

Discharge Data on Group I

	Number	*Sputum Positive*		*Sputum Negative*	
		No.	*%*	*No.*	*%*
Discharged with permission	40	3	7.8	37	92.2
Discharged without permission	7	3	42.9	4	57.1
Transferred	2	0	0	2	100
Dead	9 (15%)	0	0	0	0
Dismissed	2	1	50	1	50

TABLE XII

Discharge Data on Group II

	Number	*Sputum Positive*		*Sputum Negative*	
		No.	*%*	*No.*	*%*
Discharged with permission	32	8	25	24	75
Discharged without permission	3	1	33.3	2	66.7
Transferred	3	2	66.6	1	33.4
Dead	14 (27%)	0	0	0	0
Dismissed	0	0	0	0	0

obtaining a negative sputum on discharge, yet a gain of 14.9 per cent in those that were improved. These figures are better understood when it is seen that of the entire group studied 95.7 per cent had moderately or far advanced chest disease. There is no doubt of the value of collapse therapy, and it may be that the peak of percentage of improvement has been reached. The table shows that collapse therapy has steadily increased, and there is a gain of 25 per cent over the study last made, 1933-1938.

TABLE XIII

Discharge Data on Group III

	Number	*Sputum Positive*		*Sputum Negative*	
		No.	*%*	*No.*	*%*
Discharged with Permission	8	3	37.5	5	62.5
Discharged without permission	0	0	0	0	0
Transferred	2	1	50	1	50
Dead	17 (63%)	0	0	0	0
Dismissed	0	0	0	0	0

TABLE XIV

Discharge Data on All 139 Cases

	Number	*%*	*SPUTUM*			
			+		−	
			No.	*%*	*No.*	*%*
Discharged with permission	80	57.5	14	17.5	66	82.5
Discharged without permission	10	7.1	4	40	6	60
Transferred	7	5	3	42.8	4	57.2
Dead	40	28.8	0	0	0	0
Dismissed	2	1.6	1	50	1	50

Of the whole group only a very small percentage with positive sputums were discharged from the Sanatorium to go out to mingle with the public. The total number was 22 or 15.7 per cent. Many of those who left with permission were unsuitable for thoracic surgery. Of a few it can be said that home conditions were suitable for treatment there.

The large percentage of deaths (28.8%) indicates the seriousness of the chest disease in all cases of laryngeal involvement.

TREATMENT: MEDICAL AND SURGICAL PROCEDURES

Medical Treatment:

We feel that in those cases of incipient laryngeal disease with hoarseness and irritative cough, soothing sprays are indicated. Mono-P-chlorophenol is most often used. We also use guaiacol and menthol. Sprays are employed in the more advanced cases too, but we feel that in the first group an ounce of prevention may in some cases avert edema and ulceration. Of course, voice rest is one of the greatest aids in obtaining healing, and should be prescribed in all cases.

TABLE XV

Chest Surgery in Group	*I*	*II*	*III*
Phrenic	2	6	0
Pneumothorax	27	27	11
Pneumonolysis	6	6	2
Thoracotomy	2	2	0
Thoracoplasty	5	9	2
Bronchoscopy	1	2*	0

*One pt. 7 times
One pt. twice

TABLE XVI

Tuberculous Complications	*I*	*II*	*III*
Healed Pott's disease	1		
Tuberculous spine	1		
Enteritis	4 (6.6%)	6 (11.5%)	2 (7.4%)
Sinus of buttocks	1		
Perirectal abscess	1	1	1
Tracheobronchial tuberculosis	1	1	
Rectal fistula	1	1	2 (7.4%)
Cervical adenitis	1		
Spontaneous pneumothorax		1	
Empyema			
Pleurobronchial fistula		1	
Renal tuberculosis			3 (11%)
Miliary tuberculosis			1

Enteritis was the most frequent complication. Renal tuberculosis occurred only in Group III.

TABLE XVII

Comparative Analysis of 426 Patients Treated for Laryngeal Tuberculosis at Blue Ridge Sanatorium

	1923-1928 Percent	*1933-1938 Percent*	*(1st 3 mos., 1944) 1939-1944 Percent*
Total number of patients	1090	1139	2213
Laryngeal tuberculosis—number	136	152	139
Laryngeal tuberculosis—per cent	12.5	13.4	6.2*
Sputum positive on admission—per cent	95.6	90.1	84.5
Sputum positive on discharge—per cent	63.2	50.2	44.6**
Cauterization of larynx—number	19	29	32
Cauterization of larynx—per cent	14.0	19.0	23
Collapse therapy—number	6	75	102
Collapse therapy—per cent	4.4	49.3	73.9***
Improved on discharge—per cent	44.1	52.6	67.5****

*The incidence of laryngeal tuberculosis is 50 per cent less in this last group.

**There was a gain of 5.6 per cent in those obtaining a negative sputum on discharge. (This figure includes those that were discharged as dead.)

***There was a gain of 24.6 per cent in the use of collapse therapy.

****There was a gain of 15 per cent in improvement at discharge as the result of the increased use of collapse therapy.

TABLE XVIII

	Group I Percent	*Group II Percent*	*Group III Percent*
(1) Asymptomatic	38.3	5.7	..
(2) Hoarseness	53.3	86.5	88
(3) Cough	50	65	90
(4) Pain	3.3	15	18
(5) Soreness	5	7	9
(6) Fullness	3.2	5	12
(7) Dryness	..	1	..
(8) Congestion	1.5	7	..
(9) Aphonia	..	1.8	4
(10) Tickling	1	1	2
(11) Dysphagia	..	3.5	4.5

Surgical Treatment:

The surgical treatment of today is limited to:

(1) Galvanocauterization: This is never used in cases where the lesion is minimal, except to destroy interarytenoid tuberculomas or vegetations. It was introduced by Gruenwald of Germany in 1905. It was first used in this country by George B. Wood and Joseph B. Green, and was found to be a very potent weapon toward the relief of pain and suffering. After thorough cocainization of the larynx, several punctures are made with the electrode in the diseased sites. It is best to have the electrode at white heat. Care must be exerted not to overdo, and technique must not allow puncture of the cricoarytenoid joint or the insertion of the needle into the muscular areas, as healing may bring stenosis.

(2) Laryngoscopy: This procedure is sometimes necessary for the removal of tuberculomas. It is the consensus of opinion that the base of any tuberculous tumor should be lightly cauterized to prevent local spread.

(3) Biopsy: We have had several cases in which a differential diagnosis was made by biopsy. One patient had intrinsic tuberculosis and squamous cell carcinoma, coexisting. (Pt. did not appear in the study as he had not been discharged). Syphilis, too, is sometimes present and a biopsy with blood serology is most helpful.

(4) Removal of the Epiglottis: This is seldom done. The pain is generally abated by proper use of the cautery. However, epiglottectomy with punch or snare can be done under local anesthesia.

(5) Block of the internal branch of the superior laryngeal nerve: This was carried out twice in the present series of cases. We feel it is advisable, particularly in the painful, advanced, extrinsic cases unrelieved by cauterization. As Woodward advises, the nerve should never be resected.

(6) Tracheotomy: Tracheotomy because of edema resulting from too extensive use of the cautery hardly ever occurs, if there is a thorough understanding of the objectives and technique. Tracheotomy should never be done to put the larynx at rest, as this is not obtained. The cases that we have done were having marked laryngeal obstruction from ulceration and edema. We use the two stage operation, if time permits, as described by Woodward.

(7) Although there were no cases in this series, healing of tuberculous ulcers does cause cicatricial stenosis of the larynx, and often it is necessary to carry out some form of surgery to improve the air way. In healed cases, arytenoidectomy, as described by Kelly, has been done with a fair degree of success. A tracheal tube is generally a necessity for some time in either event.

(8) Bronchoscopy: In the treatment and diagnosis of pulmo-

nary tuberculosis, this is a very important adjunct. Aspirations, biopsies, and cauterization of tracheal tree lesions could not be done without it.

(9) Collapse therapy, as the statistics will indicate, has certainly helped us in obtaining a negative sputum, one of the first objectives toward obtaining a healed larynx.

In conclusion I would like to emphasize that the three major objectives in the treatment of tuberculosis of the chest and larynx are: first, to free the sputum of tubercle bacilli; second, rapid healing of the chest lesion with disappearance of the symptoms; and third, rehabilitation of the patient. The present study indicates that these objectives are accomplished in a greater percentage of patients than ever before. The influence of collapse therapy upon the laryngeal lesion is evidenced by the low percentage of patients having this complication. The use of electro-cautery in 23 per cent of the 139 cases presented in the last five year study, with marked relief in more than 90 per cent, emphasizes its value in our therapeutic armamentarium.

SUMMARY

In any tuberculosis institution the treatment of laryngeal tuberculosis occupies a prominent role in the therapy of this dreaded disease. The relationship of laryngeal tuberculosis to pulmonary tuberculosis is now better understood.

This is a study of 139 cases of laryngeal tuberculosis in 2213 patients discharged from the Sanatorium. The diagnosis in the early phase rests upon repeated mirror examinations of the larynx, as the voice does not become hoarse except when the cords are involved or if their approximation is interfered with by a pathological lesion in the posterior commissure. The symptoms increase with the degree of pathological involvement. The group ranged in age from the teens into the seventies. They were divided into three groups.

Group I consisted of 60 cases of incipient tuberculosis of the larynx. In these cases there was no edema or ulceration; 38.2 per cent were asymptomatic. The posterior commissure only was involved in 80 per cent of the cases. Examination of the chest revealed 8.4 per cent in the minimal group, 45 per cent moderately advanced, and 46.6 per cent far advanced. On admission 75 per cent had positive sputum, and 11.6 per cent positive sputum on discharge. Some form of chest surgery was performed on 71.6 per cent. The treatment consisted of comforting sprays and silence. The results showed 78.3 per cent of the larynges healed or improved and 76.6 per cent of the chests healed or improved.

The second group included 52 cases with slight edema and slight

ulceration. Only 5.7 per cent were asymptomatic. The pathology in this more advanced group involved the posterior commissure per se in only 19.2 per cent of the patients; 98 per cent showed moderately or far advanced chest pathology; 88 per cent had positive sputum upon admission; 21 per cent had positive sputum on discharge; and 96 per cent had some form of chest surgery. The treatment, in addition to silence and spray, consisted of galvanocautery and nerve block. The larynx improved or healed in 73 per cent of the cases; the chest improved or became arrested in 55 per cent of the cases.

The third group included 27 patients with advanced edema and ulceration of the larynx. The symptoms were marked cough, hoarseness and pain in all cases. Far advanced chest disease was present in 82 per cent, and 92.5 per cent had positive sputums. The sputum was positive on admission in 92.5 per cent and positive on discharge in 14.8 per cent. The treatment of the larynx consisted of silence, sprays, cauterization (in 66.6 per cent) and tracheotomy (1 case). The larynx healed or improved in 33.3 per cent; the chest improved in 26 per cent.

The percentage of tuberculosis of the larynx has decreased greatly during the last few years. The likely reason for this is the increased rate of early diagnosis and the early institution of thoracic surgery to obtain negative sputums. These 139 cases of laryngeal tuberculosis represent an incidence of 6.2 per cent. This is 50 per cent less than in a similar group study 1933-1938. The study further shows that there is a gain of 24.6 per cent in the use of collapse therapy. Of these 139 cases, 73.9 per cent received some form of collapse therapy as against 49.3 per cent of a similar group study 1933-1938. The death rate of 28.8 indicates the seriousness of that disease in all cases of laryngeal involvement.

The medical treatment consists of comforting spray and voice rest.

The surgical treatment consists of:

(1) Galvanocauterization in Groups II and III. The electrode is used at white heat with the larynx thoroughly cocainized.

(2) Laryngoscopy: for removal of tuberculomas.

(3) Biopsy: for differentiation between tuberculosis, carcinoma and syphilis.

(4) Block of internal branch of the superior laryngeal nerve.

(5) Tracheotomy: the two stage operation is preferable if time permits. It should never be done to put the larynx at rest.

(6) Arytenoidectomy: as described by Kelly for cicatricial stenosis.

(7) Bronchoscopy: Aspiration, biopsies, and cauterization of the tracheal tree lesions could not be done without it.

(8) Collapse therapy has certainly helped us in obtaining a negative sputum, one of the first objectives toward obtaining a healed larynx.

In conclusion I would like to emphasize that the three major objectives in the treatment of tuberculosis of the chest and larynx are: first, to free the sputum of tubercle bacilli; second, rapid healing of the chest lesion with disappearance of the symptoms; and third, rehabilitation of the patient. The present study indicates that these objectives are accomplished in a greater percentage of patients than ever before. The influence of collapse therapy upon laryngeal lesions is evident by the decreasing percentage of patients having this complication. The use of electrocauterization in 23 per cent of the 139 cases, with marked relief in more than 90 per cent, emphasizes the value in our therapeutic armamentarium.

RESUMEN

En toda institución para tuberculosos el tratamiento de la tuberculosis de la laringe ocupa un lugar prominente en la terapia de esta temida enfermedad. Se comprende hoy mejor la relación de la tuberculosis de la laringe a la tuberculosis pulmonar.

Es éste un estudio de 139 casos de tuberculosis de la laringe entre 2213 enfermos dados de alta de este Sanatorio. En el período temprano el diagnóstico depende de los repetidos exámenes de la laringe con el espejo, pues la voz no se enronquece sino cuando se encuentran invadidas las cuerdas vocales o cuando una lesión patológica en la comisura posterior impide su aproximación. Los síntomas aumentan en proporción al grado de invasión patológica. El grupo, que varió en edad de los trece a los setenta y pico de años, fue subdividido en tres grupos.

El Grupo I comprendió 60 casos de tuberculosis incipiente de la laringe. En estos casos no existía edema o ulceración y el 38.2 por ciento eran asintomáticos. En el 80 por ciento de los casos solamente estaba invadida la comisura posterior. El examen del tórax reveló que el 8.4 por ciento estaban en el período mínimo, el 45 por ciento en el moderadamente avanzado y el 46.6 por ciento en el muy avanzado. El 75 por ciento tenían esputo positivo al ingresar al Sanatorio y el 11.6 por ciento tenían esputo positivo al dárseles de alta. En el 71.6 por ciento se ejecutó alguna forma de cirugía torácica. El tratamiento consistió en el empleo de líquidos pulverizados para alivio y en guardar silencio. Los resultados revelaron que el 78.3 por ciento de las laringes se cicatrizaron o mejoraron y que el 76.6 por ciento de los tóraces se cicatrizaron o mejoraron.

El Grupo II incluyó a 52 casos con ligero edema y ligera ulcera-

ción de los que solamente el 5.7 por ciento eran asintomáticos. En este grupo más avanzado el proceso patológico invadió únicamente la comisura posterior solamente en el 19.2 por ciento de los enfermos; en el 98 por ciento la lesión torácica era moderadamente o muy avanzada; el 88 por ciento tenían esputo positivo al ingresar y el 21 por ciento al dárseles de alta; y en el 96 por ciento se aplicó alguna forma de cirugía torácica. Además del silencio y de líquidos pulverizados, el tratamiento incluyó el galvanocauterio y el bloqueo del nervio. La laringe mejoró o se cicatrizó en el 73 por ciento de los casos y mejoró o se estacionó la lesión pulmonar en el 55 por ciento de los casos.

El Grupo III incluyó a 27 enfermos con edema y ulceración laríngea avanzados. Todos los casos tenían mucha tos, ronquera y dolor. En el 82 por ciento existía neumopatía muy avanzada. El esputo era positivo en el 92.5 por ciento al ingresar, y en el 14.8 por ciento al dárseles de alta. El tratamiento de la laringe incluyó el silencio, líquidos pulverizados, cauterización (en el 66.6 por ciento) y traqueotomía (en 1 caso). La laringe se cicatrizó o mejoró en el 33.3 por ciento; el pecho mejoró en el 26 por ciento.

El porcentaje de tuberculosis de la laringe ha disminuído mucho durante los últimos años, debido probablemente al aumento en la proporción de diagnósticos tempranos y en la aplicación oportuna de la cirugía torácica para obtener la conversión del esputo. Estos 139 casos de tuberculosis de la laringe representan una frecuencia de 6.2 por ciento, o sea 50 por ciento menos que en un estudio semejante de otro grupo, llevado a cabo de 1933 a 1938. El presente estudio también revela un aumento de 24.6 por ciento en el empleo de la colapsoterapia. Entre estos 139 casos, el 73.9 por ciento recibió en alguna forma la colapsoterapia, comparado con el 49.3 por ciento en el estudio semejante de 1933 a 1938. La mortalidad de 28.8 indica la gravedad de la enfermedad en todos los casos que presentan la complicación laríngea.

El tratamiento médico consiste en el empleo de líquidos pulverizados para alivio y en el descanso de la voz.

El tratamiento quirúrgico consiste de:

(1) La Galvanocauterización en los Grupos II y III. Se usa el electrodo al rojo blanco con la laringe completamente anestesiada con cocaína.

(2) La Laringoscopia para la extracción de tuberculomas.

(3) La Biopsia para la diferenciación entre la tuberculosis, el carcinoma y la sífilis.

(4) El Bloqueo de la rama interna del nervio laríngeo superior.

(5) La Traqueotomía. La operación en dos etapas es preferible, si el tiempo lo permite. No se debe ejecutar nunca para poner la laringe al descanso.

(6) La Aritenoidectomía, como la describe Kelly, para la estenosis cicatrizal.

(7) La Broncoscopia, sin la cual sería imposible la aspiración, la biopsia y la cauterización de las lesiones del árbol traqueal.

(8) La Colapsoterapia, que indudablemente nos ha ayudado a obtener la conversión del esputo en negativo, uno de los primeros objetivos al procurar la cicatrización de la laringe.

En conclusión, desearía recalcar que los tres objetivos principales en el tratamiento de la tuberculosis pulmonar y laríngea son: primero, eliminar los bacilos tuberculosos en el esputo; segundo, la cicatrización rápida de la lesión pulmonar con la desaparición de los síntomas; y tercero, la rehabilitación del paciente. El presente estudio indica que se están alcanzando estos objetivos en un mayor porcentaje de enfermos que nunca antes. La influencia de la colapsoterapia sobre las lesiones laríngeas se pone de manifiesto en la proporción disminuyente de enfermos que sufren de esta complicación. El empleo de la electrocauterización en el 23 por ciento de los 139 casos, con decidido alivio en más del 90 por ciento, acentúa su valor en nuestro arsenal terapéutico.

REFERENCES

1 Heat, F. R. G., et al: "Promin for Treatment of Laryngeal Tuberculosis, *Lancet*, 1: 702, (June 5) 1943.
2 Donnelly, J. C.: "Pulmonary Tuberculosis Masquerading as Laryngitis," *Jour. Am. Med. Assn.*, 120: 675-679. (Oct. 31) 1942.
3 Myerson, M. C.: "Laryngeal Tuberculosis in Tuberculous Patients," *Dis. Eye, Ear, Nose & Throat*, 1: 196-198, (July) 1941.
4 Dworetzky, J. P. and Risch, O. C.: "Study of 500 Cases of Pulmonary Tuberculosis with Resume Based on 28 Years of Experience," *Ann. Otol., Rhin. & Laryng.*, 50: 745-761, (Sept.) 1941.
5 Spencer, F. R.: "Laryngeal Tuberculosis with Recent Advances in Treatment," *Ann. Otol., Rhin. & Laryng.*, 50: 735-744, (Sept.) 1941.
6 Wilson, G. E.: "Diagnosis and Treatment of Tuberculosis of the Larynx and Contiguous Areas," *Arch. Otolaryng.*, 33: 145-176, (Feb.) 1941.
7 Wood, G. B.: "Tuberculosis of Upper Respiratory Tract: Review of Literature of Past 2 Years; Summaries of Bibliographic Material Available in Field of Otolaryngology," *Arch. Otolaryng.*, 33: 861-882, (May) 1941.
8 Dworetzky, J. P.: "Etiology and Prophylaxis of Tuberculous Laryngitis," *Ann. Otol., Rhin. & Laryng.*, 23: 835-858, 1941.
9 Wood, G. B.: "The Actual Cautery in the Treatment of Localized Tuberculous Lesions," *Trans. Am. Laryng. Assn.*, ??: 181-186, 1911.
10 Gruenwald, L.: "Some Observations of the Treatment of Laryngeal Tuberculosis," *Laryngoscopy*, 20: 637-644, 1905. Cited by Greene.
11 Woodward, F. D.: Personal communications.
12 Woodward, F. D.: "Laryngeal Tuberculosis: Surgical Treatment Including Collapse Therapy," *Surg., Gyn. & Obst.*, 68: 390-394, (Feb. 15) 1939.
13 Stafford, F. B.: Personal communications.
14 Greene, J. B.: "The Electrocautery in the Treatment of Laryngeal Tuberculosis," *Ann. Otol., Rhin. & Laryng.*, 33: 193-197, (March) 1924.
15 Wood, G. B.: "Actual Cautery in the Treatment of Localized Tuberculous Lesions," *Ann. Otol., Rhin. & Laryng.*, 20: 531-535, (Sept.) 1911.

Separation Center Chest Survey

LOUIS SCHNEIDER, MAJOR, M.C.*
Mt. Vernon, New York

During the initial six months of operation of the Ft. Dix Separation Center, the first such unit to be set up by the Army, 11,033 men and women candidates for discharge from the Army were routinely examined prior to separation from service for other than physical grounds. The chest survey was performed using the photoroentgenographic method of screening, whereby a pair of 4 x 5 stereoscopic negatives was made on each individual, which was a repetition of the procedure used in most instances at the time of induction into service of these people. If, from a reading of the small films, a more exact delineation of suspected pulmonary pathology was needed, a 14 x 17 radiograph was thereupon taken. Of the total number appearing for separation, there were 9,658 enlisted men who were to be discharged because of minority, overage, hardship at home, essential civilian occupation, maladjustment to military life, etc. The 1,084 officers appeared for separation because of overage in grade, extreme hardship at home, inability to meet the requirement of their assignments, etc. The remainder or 291 were women, all except a few of whom were enlisted members of the Women's Army Corps who were being released for about the same reasons as the enlisted men. The group of women examined is too small to draw any conclusions therefrom, but it is noteworthy that among them not a single case of pulmonary pathology was discovered on the separation chest film. Following are some of the important findings in the routine chest studies of the 10,732 men examined at the Separation Center:

1. PULMONARY TUBERCULOSIS

Four minimal active cases of pulmonary tuberculosis proven by subsequent hospital study were discovered among the enlisted men up for separation from service. These were found in individuals whose induction films were negative less than two years before. This figure might be increased somewhat after further study of three additional cases suspected of having the same type of new pulmonary pathology but whose hospital records were not available at the time of the writer's transfer to duty elsewhere. In

*From the Separation Center, 1245 S. C. S. U. Ft. Dix, New Jersey, in the Second Service Command.

another man, an older officer, bilateral actively advancing pulmonary tuberculosis was found and this was some months after he had been put on limited service following treatment for an active minimal lesion discovered while he was on duty in Africa.

There were fourteen other cases of pulmonary tuberculosis who were ordered hospitalized after separation examination for further study and comparison of the discharge with the induction film. These comprised men who had significant pulmonary findings on roentgenogram greater in extent and more ominous-looking in character than we would ordinarily accept for active duty. Upon questioning, they were surprised to learn for the first time that they had any sort of pulmonary pathology. The lesions aforementioned were subsequently determined to have been present at the time of entry into service, having remained unchanged thruout the length of duty of the men concerned.

Twenty-six cases showed small healed or arrested foci of reinfection minimal pulmonary tuberculosis which one could safely assume were in need of no further observation before discharge. Even if detected at the time of induction, these men met the physical requirements for mobilization with these findings.

Comment: From this small survey, it would appear that the incidence of fresh cases of pulmonary tuberculosis in previously screened men who had seen active duty in the Army for a period of about two years is less than one in two thousand. This sample indicates too that the preponderant new pulmonary lesion found on separation examination will fortunately be the minimal one. Lee, Fein and Steinhauser[1] in 10,000 of their thoroly screened men who were re-x-rayed at examination for air crew training, found an incidence of 0.05 per cent of fresh cases of pulmonary tuberculosis. After a longer interval and dealing with a different group of individuals, Robins[2] got much higher figures in a sample survey of this size. He discovered 2.9 new cases per thousand among 8,731 apparently healthy individuals on relief rolls re-examined after an average length of time of 14.2 months elapsed since a normal chest film had been found on a previous survey. It is interesting to note that 0.4 per cent of the men appeared for separation examination with minimal arrested reinfection pulmonary tuberculosis which remained unchanged in spite of a varied Army career, many of them having been in battle zones. None of the new cases discovered on discharge were engrafted upon an old arrested lesion.

2. PNEUMONIA

Eleven cases of atypical pneumonia were seen, an incidence of about 0.1 per cent of those examined. The highest number, seven, were seen in June; there were two in September, one in May and

one in July. The typical finding was a localized fuzzy infiltration adjacent to the heart border in one or the other lower lung fields, which cleared up after a short hospital stay. In one case, however, the x-ray showed a diffuse nodular lesion predominantly in the lower half of both lungs, with a few nodules in the right upper lobe as well. This soldier was ready for separation discharge after six weeks' hospitalization. A normal bronchogram was obtained on him at the hospital, it having been done after the inflammatory process had subsided.

Six men in this whole series had come into the Separation Center with retained iodized oil in their lungs which again emphasizes the fact that bronchography is a diagnostic procedure frequently employed in Army hospitals.[3] It is worth mentioning at this time therefor, that bronchography is not without danger[4] and that it would be wise to have the patient get rid of the oil by active postural drainage after the diagnostic bronchograms have been accomplished.

3. PNEUMOCONIOSIS

Three cases of pneumoconiosis were found. This was diagnosed from the film and subsequently confirmed by the history obtained from the soldiers. The men were coal miners before entering service, one with first stage and the others with second stage pneumoconiosis. One of the latter had been working in coal mines for twelve years, till 1942 when he donned the Army uniform. None of these men had suffered any respiratory distress or any form of pulmonary or bronchial infection while on active duty. Nevertheless, silicosis as represented by strand-like and nodular shadows in the roentgenogram is ground for rejection from military service according to MR 1-9 of April 19, 1944.

4. BULLOUS EMPHYSEMA

Six cases of bullous emphysema were discovered. Five were asymptomatic and a sixth had a clinical history of non-disabling chronic bronchitis. Five had small bullae or blebs confined to an apex or the apices, while the film of one soldier, an asymptomatic young Negro, showed large bullae confined to the right upper lobe region. Bullous emphysema is very frequently an accidental asymptomatic finding in chest surveys[5,6] and the subsequent development of spontaneous pneumothorax or other pulmonary complications in individuals with these findings is open to question.

5. BRONCHIECTASIS AND BASAL FIBROSIS

Three cases of moderately severe bronchiectasis were found in enlisted men appearing for Separation Center discharge. These

were subsequently confirmed by hospital study including bronchography. The lesions were all confined to the left lower lobe and it is probable because of this location under and adjacent to the left lower heart border that the diagnosis which would have led to their rejection at induction, was missed at that time. These men admitted symptoms of long standing.

Six cases showed areas of localized firm fibrosis at one or the other lung base, and one of these was associated with small areas of plate-like atelectasis. The six men were practically asymptomatic or denied symptoms. Four were older officers and two were enlisted men. Two men were studied further in the hospital after which they were discharged thru the Center, no disabling pulmonary pathology having been found. It would appear to be more reasonable to label these cases in the Army by the term pulmonary fibrosis rather than chronic pneumonitis which some clinicians have been prone to do in the past. The latter term connotes to the individual concerned something of organic seriousness which continued observation frequently does not justify.

PULMONARY COCCIDIOIDOMYCOSIS

One case of arrested pulmonary coccidioidomycosis of the nummular type was seen in the chest film of a young white soldier up for discharge because of a very essential civilian occupation. He had developed the active disease while stationed in the San Joaquin Valley region nine months previously and had been returned to duty after hospitalization of about a month. He had been getting along well and had no complaints when he appeared for separation examination. It is felt that these arrested cases should be followed up by serial x-ray films at the Veterans' Administration Facilities at six months' intervals much as is done for an arrested case of pulmonary tuberculosis. Students of Coccidioidomycosis[7] are disinclined to believe that reactivation of an arrested case of the pulmonary phase of this ailment is a cause for serious future concern.

7. SIGNIFICANT CARDIAC FINDINGS

At induction stations, the radiologist is of frequent help to the internist by his finding and reporting of suspicious changes in the configuration and/or size of the heart or thoracic aorta. Obviously the organic cardiacs have been so thoroughly screened out before and during the period of active service, that at separation significant cardiovascular findings by x-ray were uncommon. As at the induction examination two outstanding findings of no real clinical significance were commonly observed. A prominent bulge in the region of the pulmonary conus would be seen from time

to time in youths under twenty years of age with negative history and with no abnormal physical signs on examination. A slightly widened thoracic aorta was not unusual in the young Negro with no history or clinical evidence of syphilis.

8. HILAR ADENOPATHY OTHER THAN CALCIFIED HILAR NODES

One case of moderately dense knob-like shadows in the root regions was found in the chest film of a soldier who at 45 was being discharged for overage. This adenopathy had not changed in extent or character since induction a year and a half before. He had been stationed abroad and had no signs or symptoms. His past history had been negative.

Much larger and more dense and discrete knob-like hilar shadows were seen in the routine film of a Negro soldier of 22 who was about to leave the service. The case was suspicious of Boeck's Sarcoid and the man was hospitalized but no further data are available at this time.

9. MISCELLANEOUS FINDINGS

a) Large sub-sternal thyroid, non-toxic, in an officer who had knowledge that this condition existed when he reported for duty. There was no change in the benignity or size of the enlarged thyroid at the end of service here and abroad.

b) One film showed a large calcified pleural plaque in the right lower lobe region.

c) Two cases with wide interlobar densities which probably represented organized interlobar exudate following empyema in this region. These men were asymptomatic at the time of discharge.

SUMMARY

1) Like other case-finding chest film mass studies of apparently normal individuals, routine photoroentgenograms done at a separation center are fruitful of uncovering new cases of pulmonary tuberculosis.

2) The majority of such cases found are minimal lesions.

3) Many cases of arrested minimal pulmonary tuberculosis have withstood varied periods of Army life without reactivation of the original lesion.

4) Because of the peculiar location in relation to the heart, cases of left lower lobe bronchiectasis may be easily missed in a survey.

RESUMEN

1) Lo mismo que otros estudios colectivos de descubrimiento de casos entre sujetos aparentemente normales mediante peliculas

torácicas, las roentgenfotografías sistemáticas tomadas en un centro de separación del servicio militar dan por resultado el poner al descubierto nuevos casos de tuberculosis pulmonar.

2) La mayor parte de los casos descubiertos son de lesiones mínimas.

3) Muchos casos de tuberculosis pulmonar mínima estacionada han resistido diferentes períodos de la vida militar sin reactivar la lesión primitiva.

4) Debido a su localización peculiar con relación al corazón, es fácil pasar por alto en una encuesta casos de bronquiectasia del lóbulo inferior izquierdo.

REFERENCES

1 Lee, R. E., Fine, A., and Steinhausen, T. B.: "Experiences with Photoroentgen Methods in Chest Surveys," *South. M. J.*, 37: 217 (Apr.), 1944.
2 Robins, A. B.: "The Development of Tuberculosis in the Apparently Healthy Adult," *Am. Rev. Tuberc.*, 47: 1 (Jan.), 1943.
3 "Notes on Certain Diseases of the Chest," *Technical Medical Bulletin No. 69*, War Department, Washington, D. C., July 22, 1944.
4 Schneider, L., and Segal, J.: "Bronchography with Iodized Oil not without Danger," *Am. Rev. Tuberc.*, 35: 590 (Apr.), 1937.
5 Ornstein, G. G., and Lercher, L.: "Spontaneous Pneumothorax in the Apparently Healthy Individual," *Quart. Bull. Sea View Hosp.*, 7: 149 (Apr.), 1942.
6 Schneider, L., and Reissman, I. I.: "Idiopathic Spontaneous Pneumothorax—History of 100 Unselected Cases," *Radiology*, 44: 5 (May), 1945.
7 Smith, Edward: Personal Communication.

The Chest Specialist: His Training and Services*

MILTON S. LLOYD, M.D., F.C.C.P.
New York, New York

In the realm of diagnosis and treatment of chest diseases a peculiar situation exists today. It is fairly well represented by the school-boys definition of a skeleton: "A man with the outside off and the inside out." Since the turn of the present century, an entire new specialty has been developed in this field. It includes x-ray, pneumothorax, bronchoscopy and esophagoscopy, bronchography, thoracoscopy, closed pneumonolysis and surgery.

It is natural that these specialized methods should have been developed from the older branches of medicine. Radiography, of course, resulted from the work of the x-ray specialist. But, the chest specialist has long since realized his helplessness without the exploitation of this field himself. He, therefore, takes and interprets his own x-rays as an expert. Pneumothorax was first advocated in this country by a surgeon. But, again, this method of therapy was turned over to the chest specialist as an essential part of his armament. Bronchoscopy—or bronchoesophagology, to give it its up-to-date title—was a normal extension of the work of the laryngologist. This specialty, devoted exclusively to the diagnosis and treatment of diseases of the chest, has been broadened and perfected to a point at which it may now be said that, without it, the chest specialist cannot adequately cover his field. At least fifty per cent of the physical findings in lung diseases are to be found inside the bronchial tubes, to say nothing of the fact that the mediastinum contains the esophagus. Its importance as an adjunct to therapy has also increased until it can claim a place almost equal to the x-ray, even in the treatment of pulmonary tuberculosis. Thoracic surgery has grown with equal speed. Here, also, the surgeon without bronchoscopy is finding increasing difficulty in the close correlation of ideas and observations necessary to the full perfection of his work. The time is now ripe for the bringing together of these scattered parts to permit them to coordinate and implement each other under single direction.

The existing alignment of thoracic specialties is a result of the accidental chronology of their development and bears no relation to their most efficient application or to demand in practice. The

*Presented before the Conference of College Chapter Officials, Tenth Annual Meeting, American College of Chest Physicians, Chicago, Illinois, June 12, 1944.

chest specialist, it is true, without their full exploitation, suffers from an important handicap in his work. But this is trivial compared with the fact that, for the same reason, the public is, to a great extent, deprived of his services. The avowed intent of the medical profession is to render the greatest service to the greatest number at the lowest possible cost. Nowhere else in the entire field of medical practice, however, does the profession fall so far short of this ideal as in the realm of chest diseases. Outside of the large urban centers, the average American hospital has no service whatsoever in chest diseases. This is important at a time when private enterprise in medical practice is struggling for its very existence against external supervision by governmental bureaus or by self-appointed representatives of industrial, labor or population groups.

The proponents of state medicine are quick to employ our failures against us. In the field of chest diseases, one of the strongest flanking movements is now being pressed with vigor from many sources. This failure on our part may be assigned to two causes. The first is that, in a community served by the average hospital, the physician who confines his activity to medical chest diagnosis and pneumothorax cannot support himself. He is forced to engage in general practice. He quickly loses his identity as a specialist and, in fact, ceases to be one. This leaves the hospital to serve the public as best it can with a chest specialist retiring into failure because of arbitrary restrictions and the otolaryngologist and the general surgeon attempting to occupy an over-extended field in which few, if any, can achieve competence. This is not intended to convey any suggestion of lack of ability in either of these specialised groups. It is intended to indicate, however, that there is no substitute for a sound clinical and pathological understanding of chest conditions as a groundwork for the success of any endoscopic or surgical procedure carried out upon the chest. I wish to emphasize, also, that these remarks are not intended to apply to the large cities where an abundance of clinical material has permitted men of international calibre to arise by devoting themselves exclusively to that portion of chest diseases covered by their respective and recognized specialties. It would be selfish and disrespectful on my part not to offer a grateful tribute to many of these men. To Chevalier Jackson, to Gabriel Tucker and to Louis Clerf I am indebted beyond my capacity ever to repay. From them, I learned as much about chest pathology as from any other single source, at the same time that I received hospitality and kindness outside the strictly professional field. The same may be said of Honan, Coryllos, and Davidson. The interest on such a debt accrues with the passage of time and, like many another

debtor, I find that to amortize the original sum, I must ask for even more.

The second cause of failure derives from the lack of instruction to medical students in pulmonary tuberculosis and from the presently accepted practice of shunting all tuberculosis cases out of the regular avenues of medical supervision (Fig. 8). In this way, the interne, and eventually the general practitioner, is deprived of every opportunity of studying pulmonary tuberculosis. Osler said, "Know syphilis and you know medicine." In the same way, it may be said, "Know tuberculosis and you know chest diseases" and, conversely, "If you don't know tuberculosis, you don't know chests." But the general hospital, which always has served and always will serve the public, and the family physician, who always has been and always will be the backbone of the medical profession, have been completely circumvented and even ignored. If the population of the United States is divided on the basis of service to tuberculosis patients, we find that approximately thirty five million may conveniently secure such care; the remaining one hundred million cannot. These figures were arrived at after consultation with two statisticians who do not wish to be quoted. Their accuracy may be disputed and the borderline may be shifted slightly up or down, but the truth which they represent remains the same. It is frequently necessary for the patient to travel one hundred miles for such a simple procedure as a pneumothorax refill. This mistake has been carried so far that the antituberculosis workers now project their ideas completely over the heads of the medical profession and propose by government or community sponsored schemes to x-ray the chests of the entire populace. They fail to recognize that air-power alone cannot conquer the enemy, but that he must be destroyed, and his territory occupied, by a trained army on the ground. This army is represented by the family physician. The brilliant possibilities of this effort can never be realised until every general practitioner is enlisted as a representative in the field. Unfortunately, many physicians, including some leaders in thought in the tuberculosis field, have been deluded by propaganda into believing that such a program is a solution in itself. On the other hand, the claim of the modern antituberculosis program to have had any influence at all upon morbidity and mortality from the disease is disputed by the promoters of better housing and improved living standards.

The mistakes and shortcomings which I have pointed out in previous paragraphs are not deliberate. If they were, they would be much more serious. But, at the present time, a deliberate attempt of real seriousness is being made to cast the future in the mold of the past. In the happy days before the war, a friend of mine

spent his annual vacation in the lake region of northern Ontario. Only his love of the great outdoors overcame his objection to the dirt roads which continued beyond the pavement as he neared his journey's end. But he posted a sign at the junction of the good road with the bad. It read, "Choose your rut carefully. You'll be in it for the next hundred miles." The medical profession also gets into such ruts, but I hope that the end of the "hundred miles" may be near at hand so far as the specialty of chest diseases is concerned. In this field an attempt is being made to throw a cordon around the chest specialist and to prevent him from developing his own specialty to the full advantage of himself and his patient.

Let us examine the fate of the modern chest specialist who seeks to follow this rule by making use of all available means for the patient's service. The American Broncho-esophagological Association denies him admission because he is interested more in bronchoscopy than in diseases of the ear or the throat. The Advisory Board for Medical Specialties has no place for him. He may qualify in bronchoscopy by becoming an ear, nose and throat specialist. He may meet their requirements in thoracic surgery by becoming an abdominal surgeon. If he wishes recognition in tuberculosis, he must give up his observations upon tuberculosis of the bronchi; he must cease the performance of Jacobaeus operations and thoracoplasties and begin the specialised study of diseases of the stomach, liver and kidneys in order first to obtain their diploma in internal medicine. This last requirement has set a pattern for future study by which the newborn specialist, with his first breath of life, provides for his own extinction. He is committed in advance to the elimination of pulmonary tuberculosis. But the accomplishment of this much-to-be-desired objective would, at the same time, eliminate the specialty itself. The prospect is bewildering. I am sure that, in the entire history of medicine, such a ridiculous situation never before has prevailed. What would happen to urology if that specialty were suddenly divided into radiography, bladder irrigations, cystoscopy and surgery, each under the control of a different specialist? The American College of Surgeons states that they "do not recognize thoracic surgery as a specialty." I am sure that this body would perform a greater service to the art of surgery, as well as to the public welfare, if they imposed the same requirement upon the abdominal surgeon in respect to thoracic surgery as they place upon the thoracic surgeon in respect to abdominal surgery.

I believe that these groups have an important place in the development and maintenance of high standards in medical practice. They deserve the adherence and support of all interested phys-

icians. It would be unfortunate, however, if their good intentions served to retard, rather than advance scientific progress.

In seeking a departure from this present tendency, there is nothing revolutionary or even counter-revolutionary in my mind. I wish to see, on the contrary, a return to the fundamentals of medical practice which have carried it successfully through many centuries of history. Sydenham was, at one time, looked upon as a "violent revolutionary," but, today, Castiglioni appraises him as "a common sense practitioner who understood that . . . . the chief aim of the physician should be to make himself useful to the patient."

I bring these matters to your attention because I think they are of concern to everyone who contemplates entering upon the specialty of chest diseases. Throughout this country, at the present time, young men and women are being trained in sanatoria and hospitals to become the phthisiologists and thoracic surgeons of the future. Wherever I have visited, I have found the uniform demand among them to be permitted to learn bronchoscopy. Just as uniformly, however, they have been denied this privilege. They are forced to restrict themselves in their ability to serve their patients by conforming to rules of practice which derive from origins not concerned with the patients' needs.

The visitor to Rome in normal times was frequently astonished to find, among the greatest paintings in Italian art, several productions of Michael Angelo. His name was familiar as the world's greatest sculptor. But Michael Angelo was a painter also. He had learned the value of fundamentals in his chosen field—he did both bronchoscopy and surgery.

During that epoch, the Pope was a great temporal, as well as spiritual power. We have had plenty of recent evidence that he still is, but, in those days, the world knew it in advance. And one day, the Pope wanted a paint job done. He called in a young man who had been recommended to him and he said to this not very imposing individual, "And what can you do?" The man picked up a piece of chalk and drew a perfect circle. That is a difficult feat. The Pope was impressed and gave him the job. But every day following that, he heckled and annoyed this poor workman as he struggled upside down upon the scaffolding, below a curved and difficult ceiling. Finally, the man could stand it no longer and he hurled his paint pot at the Pope's head! What did the Pope do? He retired before this man who could draw a perfect circle and Michael Angelo completed the Cistine Chapel! Certainly, it is not necessary for the chest specialist to throw a paint pot at the Pope's head, but, at least, he must have the courage to do his work according to the way in which he knows it ought to be done.

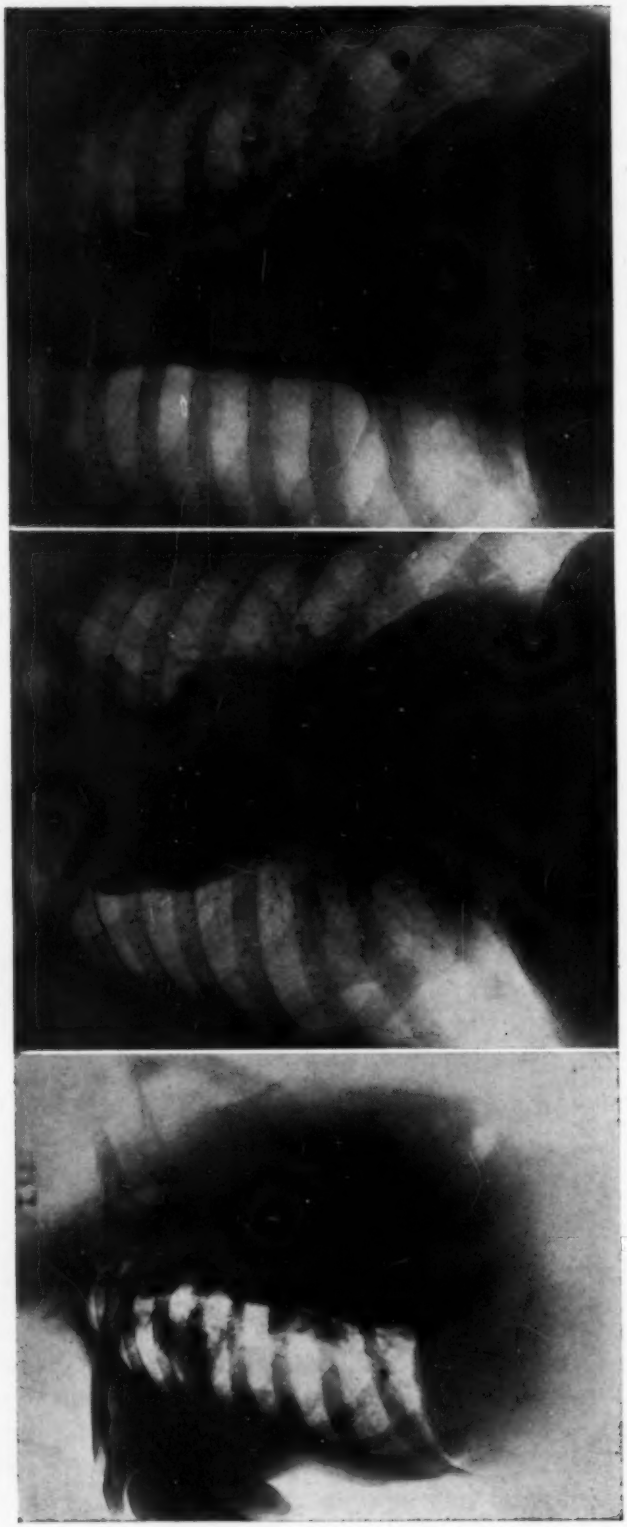

Figure 3

Figure 2

Figure 1

I would like to present this problem from the patient's point of view by showing a few cases in which the patient was exposed to inconvenience, or even improper treatment, because of bad medical organization.

CASE REPORTS

Case 1: E. A., white woman, age 44, who has, as you see (Fig. 1), a bilateral tuberculosis with a pneumothorax on the right side and a total atelectasis of the left lung following a two-stage thoracoplasty. She is, therefore, in need of the services of three specialists: one for pneumothorax, one for bronchoscopy and one for surgery. This is a difficult burden for the private case, even if the average general hospital were prepared, as it should be, to render such services.

Case 2: B. G., white man, age 53, suffering from aneurysm of the innominate artery, with compression of the trachea. Bronchoscopy was recommended in the belief that it might be a primary bronchogenic carcinoma. Pneumothorax on the right side, however, demonstrated that the mass was extrapulmonary (Fig. 2). A bronchoscopy was done then, but, naturally, without biopsy. I show this case, however, because I have been present on two occasions when death from hemorrhage followed a bronchoscopic biopsy under similar conditions.

Case 3: W. S., white male, age 57, had suffered from cough and dyspnoea for about seven years. He had been subjected to bronchoscopy because the roentgenologist reported a collapsed right lung. He was, however, suffering from a chronic spontaneous pneumothorax (Fig. 3).

Case 4: A. B., white woman, age 53, who swallowed a perforating foreign body while eating chicken soup (Fig. 4). Twenty-four hours later, there was evidence of an early mediastinitis (Fig. 5). Emergency

Figure 4

Figure 5

anterior mediastinotomy was necessary as a lifesaving measure. The management of such a case requires the continuous attendance of a well trained chest specialist.

Case 5: C. H., white male, age 60, had suffered from cough of three months' duration, hemoptysis on several occasions (Fig. 6). He was seen by two chest consultants, one of whom diagnosed fibrosis of the left lower lobe. The other stated that he would rather let his patient die than to cause him to submit to a bronchoscopy. One year later (Fig. 7), carcinoma of the bronchus was diagnosed by bronchoscopy, but was then inoperable. That was ten years ago. But the early diagnosis of carcinoma of the lung is no nearer now than it was then. I do not believe that it will ever be brought any nearer until the chest consultant is himself an experienced bronchoscopist and the bronchoscopist is equally familiar with diseases of the lungs.

Illustrative cases of this kind could be repeated indefinitely but the subject does not need to be labored. It is enough to demonstrate that progress in the chest specialist's field is seriously retarded by the confusion in action which inevitably follows confusion in organization.

In my opinion, therefore, steps should be initiated to remedy the present situation by a gradual evolutionary movement, to insure that the coming generation of chest specialists shall not be deprived of freedom of activity in their own field. I would like, therefore, to lay before the Chapter Officials the following recommendations of concern particularly to chest specialists:

1. A return to the Hippocratic principle in medical practice, where the interest and welfare of the patient take precedence over political or emotional considerations.

2. The broadening of the specialty of chest diseases to compre-

Figure 6

Figure 7

hend all methods for diagnosis or treatment of diseases of the chest. This field might be given a new name—pneumonology, for example. It should be recognized as a specialty both by the American College of Surgeons and the Advisory Board for Medical Specialties.

3. The devotion of an effort, in medical schools, to the teaching of chest diseases—particularly pulmonary tuberculosis—which is as great as the assignment in the teaching of other equally important specialties or diseases.

4. The resumption, upon a reasonable scale, of the handling of tuberculosis cases through the accepted channels of medical practice, and the encouragement of general hospitals to devote from 3 to 4 per cent of their bed capacity to the treatment of pulmonary tuberculosis cases requiring collapse therapy.

5. Hospitals and sanatoria now charged with training the rising generation of chest specialists should be urged to provide these men and women with adequate experience in all branches of chest work.

6. The general hospital should be expected to train its internes to be as capable in the diagnosis and treatment of pulmonary tuberculosis as in other diseases. The general practitioner should be given suitable responsibility in the diagnosis and treatment of pulmonary tuberculosis.

7. The general hospital should be encouraged to establish and develop departments of Pneumonology so that the chest specialist

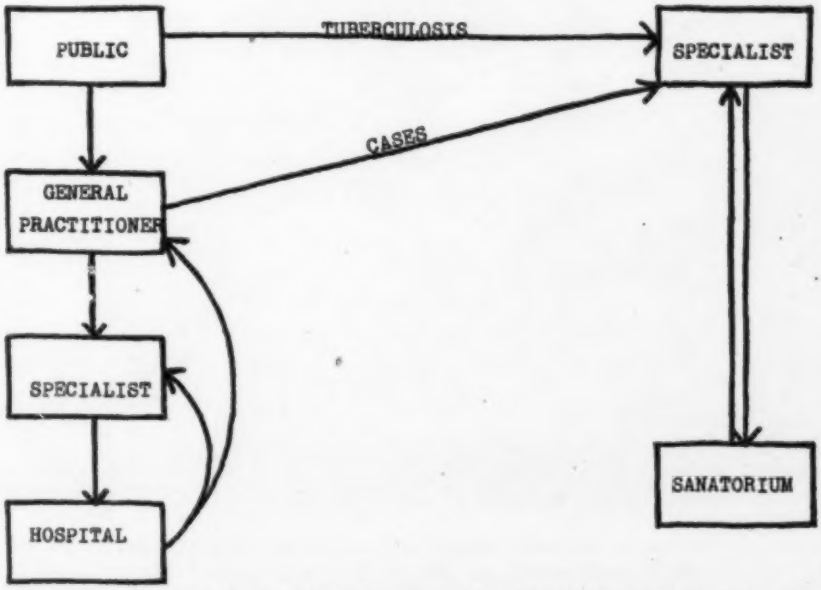

Figure 8: Diagram illustrating the route by which tuberculosis cases are shunted out of the usual channels of medical practice and the general practitioner is excluded from this field of work.

may be able to render services and to enjoy security equal to that of other specialties (Figs. 8 and 9).

8. All antituberculosis workers should seek to coordinate and unify their efforts in order that they may be able to work together as a team. This implies that the medical profession as a whole be prepared to play its proper part as a member of the team. It seems obvious to me that "early diagnosis campaigns" can never be effective without the participation—even the leadership—of the family physician.

As I said before, there is no radical thought behind any of these suggestions. Such a program, which is nothing more than a natural result of modern scientific progress, could be carried out without interfering in any way with the work of present practitioners who were trained under a different scheme. The generation of chest specialists now being trained could be, and undoubtedly would be, absorbed entirely by the general hospitals in small

Figure 9: The young chest specialist is seen attempting to ascend the difficult path toward thoracic surgery. One of his legs is pulled off and entangled in the wires of the bronchoscope. The other is similarly retained in the tubing of a pneumothorax instrument. His left arm, representing thoracoplasty, is detached and tied to the sanatorium. He still bears a look of exhausted determination and, with the right hand, raises the banner "Excelsior".

communities where they would find a now unexploited field and where their services are badly needed. In this way, the profession itself would carry out one of the most essential "relocation" movements without having such action thrust upon them by external dictation.

I am sure that, when this subject is viewed in its broader implications, our colleagues in otolaryngology and general surgery will bring to our support that sum of more which we must add to our present great and forever irrepayable debt. I am also sure that the American College of Chest Physicians, by taking the lead in this movement, would help in making one of the greatest contributions of the century to practical medicine.

"Nothing is permanent but change." There is nothing so futile as opposition to the inevitable:

> "The old order changeth, yielding place to new
> And God fulfills himself in many ways
> Lest one good custom should corrupt the world."

At a time when many of our greatest destinies are being changed before our very eyes, I should like to offer this verse as the watchword in your deliberations:

> There is no power by which a man
> Can bend a river from its plan
> To reach the sea.
> What tho' he cast athwart its face
> Great barriers from place to place,
> The river rises in its strength
> And calmly dominates at length
> Its destiny.
>
> Man is a river flowing through
> The vale of time into the blue
> Eternity.
> And he who keeps his purpose true,
> Within its course, as rivers do,
> Shall, spite of drought and spite of flood,
> Change wrong to right, change bad to good
> And slave to free.

SUMMARY

1. Since the turn of the century, the chest specialist's field has been developed and perfected to a high degree.

2. This field now comprehends a number of methods and procedures requiring specialized training such as x-ray, pneumothorax, bronchoscopy and surgery.

3. All these methods are closely interrelated and knowledge in any one of them enhances ability in the others.

4. At present, students in this specialty are prevented from reaching a maximum perfection in their work by regulations and restrictions carried over from the past. These regulations also force students in other specialties, as well as their teachers, to devote unnecessary time to fields of training which will never be used in practice.

5. It is recommended that the chest specialist be permitted the same freedom of action in the exploitation of his own field as is enjoyed by other specialties.

6. Such a course would permit the chest specialist to stand upon his own resources and to carry the benefits of his training to the general populace through existing hospitals by creating new outlets for his services.

RESUMEN

1. Desde a principios de este siglo, el campo del especialista de enfermedades del pecho ha sido desarrollado y perfeccionado en alto grado.

2. Este campo abarca ahora varios métodos y procedimientos que requieren adiestramiento especial, tales como la roentgenología, el neumotórax, la broncoscopia y la cirugía.

3. Todos estos métodos están íntimamente relacionados unos con otros y la pericia en cualquiera de ellos aumenta la habilidad en los demás.

4. Al presente, se les impide a los estudiantes de esta especialidad alcanzar una perfección máxima en su labor debido a reglamentos y restricciones heredados del pasado. Estos reglamentos también obligan a estudiantes de otras especialidades, y a sus preceptores, a dedicar tiempo innecesario a campos de adiestramiento que no usarán nunca en la práctica.

5. Se recomienda que se le permita al especialista de enfermedades del pecho la misma libertad de acción en la explotación de su campo propio de que disfrutan otras especialidades.

6. Tal proceder le permitiría al especialista de enfermedades del pecho valerse de sus propios recursos y llevar los beneficios de su habilidad a la población en general por conducto de los hospitales existentes mediante la creación de nuevas oportunidades para sus servicios.

EDITORIAL

THE IMPORTANCE OF SPECIFIC INFORMATION IN DIAGNOSIS

In 1928 when an internationally known pathologist was asked about the etiology of pulmonary calcifications, he replied that more than 99 per cent are due to tuberculosis. It was early determined that calcium is often deposited in primary tuberculous lesions, after which apparently most pathologists, upon seeing calcified pulmonary or regional lymph node lesions, diagnosed them as tuberculosis without resorting to microscopic inspection or culture and inoculation methods. When roentgenology came into being, those who made inspections of images on the fluoroscopic screen and roentgenogram almost without exception "diagnosed" shadows thought to represent calcification as primary tuberculosis.

It is difficult to understand why so many physicians accepted the erroneous belief that calcium deposits in the lungs and regional lymph nodes are pathognomonic of tuberculosis, since it has long been known that the deposition of calcium is not a specific process. During the past decade heated controversies, almost bitter at times, were waged between those who maintained that calcium deposits always represent tuberculous lesions and those who demanded more specific evidence. The tuberculin test was occasionally condemned by those who saw evidence of calcium deposits on x-ray films of the chests of nonreactors to tuberculin. Thus, x-ray shadows, which are never pathognomonic, were allowed to supersede the accurate and highly specific tuberculin test.

In 1936 Frimann-Dahl and Waaler made post-mortem examinations of 200 bodies with ante-mortem x-ray films of their chests available. They identified the primary tuberculosis complex in forty-two. In forty-eight cases there were ossifications and calcifications and thickened pleura which were nontuberculous in origin but which had produced shadows so frequently misinterpreted as parts of primary complexes.

In 1942 Aronson et. al. reported a close correlation between the presence of calcium deposits in the lungs and sensitivity to coccidioidin, suggesting that calcified pulmonary nodules may be due to infection with C. immitis.

Further observation proved that in certain areas, such as California and Arizona, a mild form of coccidioidomycosis develops in man which usually causes no significant symptom. The primary

lesions in the lungs and regional lymph nodes throughout their development, including deposition of calcium, appear identical with those of the primary tuberculosis complex. There is no way to differentiate between the lesions of these diseases except with the coccidioidin and tuberculin tests and by recovering the organisms (usually at post-mortem following death from other causes).

While examining a large number of student nurses for tuberculosis, Palmer found that evidence of calcium deposits in the chest occurred with considerable frequency in the central eastern half of the United States, the highest percentage being in Kansas City. The majority of these students were nonreactors to tuberculin. The histoplasmin test was administered to 3,105 such students and the incidence of reactors corresponded with that of calcifications in the chest, reaching its height in Kansas City, Kansas, with 54.0 and in Kansas City, Missouri, with 65.8 per cent.

A total of 294 showed evidence of pulmonary calcification, 21.4 per cent of whom reacted to tuberculin. Of the remaining 231, a definite or doubtful reaction to histoplasmin was present in 206. Only 25 failed to react to either tuberculin or histoplasmin. Among nurses reacting only to tuberculin, 10.4 per cent showed evidence of pulmonary calcifications, while among the histoplasmin reactors, 31.1 per cent showed such evidence. Of the 2,141 nonreactors to both histoplasmin and tuberculin, calcification was reported in only 1.2 per cent.

Although histoplasmosis caused by Histoplasma capsulatum was thought to be universally fatal, Palmer's work indicates that infection with this or an immunologically related organism also results in a prevalent, mild disease which can be diagnosed only by the histoplasmin test or recovery of the organism. Palmer is cognizant of the possibility that histoplasmin may not be entirely specific for histoplasma infection but may elicit the presence of some other closely related fungus infections, inasmuch as cross-reactions are frequent with such testing material as blastomycin, as shown by Emmons et al. Moreover, it may be possible that in some individuals there is co-existence of two or more kinds of fungus infections.

Christie and Peterson reported a fatal case in a five-month old infant who reacted to histoplasmin and from whom cultures of Histoplasma capsulatum were grown from bone marrow and blood. In an infant of ten months who died from another condition, cultures of Histoplasma capsulatum were grown from hilum lymph nodes and partially calcified lesions in the lower lobe of each lung. This was believed to represent a case of benign histoplasmosis. The histoplasmin and tuberculin tests were then administered to 181 children from middle Tennessee, of whom 24.9 per cent reacted

to tuberculin, and 73.5 per cent to histoplasmin. Nearly twice as many (43.6 per cent) histoplasmin reactors showed evidence of pulmonary calcification as tuberculin reactors (24.9 per cent).

Certainly not all persons who develop primary tuberculosis, coccidioidomycosis or histoplasmosis present demonstrable deposits of calcium by x-ray inspection. In tuberculosis it is rare to find as many as 20 per cent who actually have primary complexes with any evidence whatsoever by x-ray. The fact that shadows are not seen on x-ray films does not constitute proof of the absence of tuberculosis, coccidioidomycosis or histoplasmosis. Doubtless there are cases which do not calcify. In others, the deposits are so small or so located that they are not seen in the 75 per cent of the lung visualized on an ordinary x-ray film.

The diagnosis of any calcified or non-calcified pulmonary or pleural lesion, regardless of its location, size, or the appearance of the shadow it casts on the x-ray film, is dependent upon specific evidence. The physician assumes tremendous responsibility when he labels any lesion or condition of the lungs or pleura as tuberculous in the absence of a tuberculin reaction or the actual recovery of tubercle bacilli.

J. A. M.

REFERENCES

Aronson, J. D., Saylor, R. M. and Parr, E. I.: "Relationship of Coccidioidomycosis to Calcified Pulmonary Nodules," *Arch. Path.*, 34: 31, 1942.

Christie, A. and Peterson, J. C.: "Pulmonary Calcifications in Negative Reactors to Tuberculin," *Am. J. Pub. Health*, 35: 1131, 1945.

Crimm, P. D. and Short, D. M.: "Tuberculin Anergy in Cases with Pulmonary Calcifications," *Am. Rev. Tuberc.*, 39: 64, 1939.

Emmons, C. W., Olson, B. J. and Eldridge, W. W.: "Studies of the Role of Fungi in Pulmonary Disease. 1. Cross Reactions of Histoplasmin," *Pub. Health Reports*, 60: 1383, 1945.

Lumsden, L. I., Dearing, W. P. and Brown, R. A.: "Questionable Value of Skin Testing as a Means of Establishing an Epidemiological Index of Tuberculous Infection," *Am. J. Pub. Health*, 29: 25, 1939.

Myers, J. A.: "The Detection of Tuberculous Infection," *J.A.M.A.*, 112: 1904, 1939.

Nelson, W. E., Mitchell, A. G. and Brown, E. W.: "The Intracutaneous Tuberculin Reaction Associated with Calcified Intrathoracic Lesions," *Am. Rev. Tuberc.*, 37: 311, 1938.

Palmer, Carroll E.: "Nontuberculous Pulmonary Calcification and Sensitivity to Histoplasmin," *Pub. Health Repts.*, 60: 513, 1945.

Sayers, R. R. and Meriwether, F. V.: "Miliary Lung Disease Due to Unknown Cause," *Trans. Natl. Tuberc. Assn.*, p. 85, 1930.

Smith, C. E.: "Coccidioidomycosis," *Med. Clinics N. America*, 27: 790, 1943.

12th Annual Meeting of the College

Hotel Sir Francis Drake, San Francisco, California

June 29-30, July 1-2, 1946

Annual Meeting, American Medical Association

San Francisco, July 1-5, 1946

San Francisco will be host this year to the American Medical Association and the American College of Chest Physicians. A number of other medical specialty societies are also planning to hold meetings in San Francisco. Many physicians who have spent long hours in connection with their busy practices are looking forward to attending these meetings. Physicians who, because of military duties, have not been able to attend medical meetings during the war years will also want to attend the meetings in San Francisco this year.

From present indications it would seem that there will not be a sufficient number of hotel rooms to take care of every physician who plans on attending the meetings. The housing committees of the College and of the American Medical Association will, however, be pleased to do everything in their power to see that accommodations are found for as many physicians as possible. If you plan on attending the meeting we urge that you obtain your hotel reservations *at once*. For further information address:

Dr. Harry C. Warren, *Chairman*, Housing Committee
American College of Chest Physicians
384 Post Street, San Francisco 8, California

or:

Dr. M. Laurence Montgomery, *Chairman*, Housing Committee
American Medical Association
200 Civic Auditorium, San Francisco 2, California

Preliminary Program

Evidence of the Efficacy of Streptomycin in Experimental Tuberculosis
Dr. W. H. Feldman and Dr. H. C. Hinshaw, Rochester, Minn., (by invitation)

Clinical Use of Streptomycin in the Treatment of Tuberculosis
Dr. K. H. Pfuetze, F.C.C.P., Cannon Falls, Minn., and
Dr. H. C. Hinshaw, Rochester, Minn. (by invitation)

Immobilization of Both Lungs by the Equalizing Pressure Chamber in Pulmonary Tuberculosis
Dr. Alvan L. Barach, New York, N. Y. (by invitation)

Mediastinal Tumors
Dr. Brian Blades, F.C.C.P., Washington, D. C.

Surgical Treatment of Tuberculoma of the Lung
Col. John B. Grow, F.C.C.P., Denver, Colorado

Thymectomy in the Treatment of Myasthenia Gravis
Dr. Ralph Adams, F.C.C.P., Boston, Mass.

Reconditioning Following Thoracic Surgery in the Tuberculous
Major Ora L. Huddleston, Denver, Colorado (by invitation)

Inhalation of Carbon Dioxide for the Management of Cough
Dr. Andrew L. Banyai, F.C.C.P., Milwaukee, Wisc.

Observations on Cystic Emphysema of the Lung
Major E. Korol, Batavia, New York (by invitation)

Inhalation Therapy in Bronchial Injections
Dr. Edwin R. Levine, F.C.C.P., Chicago, Illinois

Experiences in the Pacific Theatre with Air Evacuation of Patients Suffering from Wounds of the Chest
Captain Leon B. Carson, Norfolk, Virginia (by invitation)

Treatment of Complications Arising During the Course of Collapse Therapy for Pulmonary Tuberculosis
Comdr. Morton R. Gibbons, Corona, California

Tuberculous Veteran, Plans for His Future
Col. Roy A. Wolford, F.C.C.P., Washington, D. C.

The Significance of the Time Element in Tuberculous Infiltrations
Dr. Herbert A. Burns, F.C.C.P., St. Paul, Minn.

Hazards of Bovine Tuberculosis as a Matter of Public Health Significance, and a Potential Human Lung Infection with the Bovine Tubercle Bacilli
Dr. H. M. O'Rear, Sacramento, California (by invitation)

Patent Ductus Arteriosis; Recent Surgical Status
Dr. J. W. Nixon, F.C.C.P., San Antonio, Texas

Mass Radiography in Tuberculosis Control
Dr. Herman E. Hilleboe, F.C.C.P., Washington, D. C.

A Critical Study of Pneumoperitoneum and Phrenic Nerve Crush in Pulmonary Tuberculosis
Dr. Allan Hurst, F.C.C.P., Dr. H. M. Maier and
Dr. Ralph Dwork, Denver, Colorado

Treatment of Tension Cavities with Pneumothorax
Dr. I. D. Bobrowitz, F.C.C.P., Otisville, New York

Motion pictures of the latest advances in the treatment of chest diseases will be shown on each day of the meeting.

Scientific Program Committee
Dr. Minas Joannides, *Chairman*
Major General S. U. Marietta
Colonel John B. Grow

"Information Please in Medicine"

"Information Please in Medicine" will again be presented as a feature of the College meeting. A group of recognized experts in medicine, surgery, pathology and roentgenology will be on hand to answer your questions. All questions should be sent to the Executive Offices of the College at Chicago well in advance of the meeting. The questions which are accepted will be published with the answers in "Diseases of the Chest" following the meeting.

Luncheon Conferences

The following luncheon conferences will be held during the meeting.

Conference of Tuberculosis Committees

Dr. Herman E. Hilleboe, Chief, Tuberculosis Control Division, U. S. Public Health Service, Washington, D. C., will address the Conference of Tuberculosis Committees of the state and county medical societies. He will talk on "State and Local Problems in Tuberculosis Control." A general discussion will follow Dr. Hilleboe's talk and a National Conference of Tuberculosis Committees will be organized. This Conference is to be sponsored by the National Council of Tuberculosis Committees of the College and will meet at the time of the annual meeting of the College.

Dr. James H. Stygall, Indianapolis, Indiana, Chairman of the Council will preside at the Conference.

Conference of Medical Directors and Superintendents

Dr. J. Winthrop Peabody, Washington, D. C., Past-President of the College and Professor of Diseases of the Respiratory System, Georgetown University School of Medicine, is to be the speaker at the Conference of Medical Directors and Superintendents of Tuberculosis Hospitals and Sanatoria. This Conference will be sponsored by the Council on Sanatorium Standards and Administration of the College under the Chairmanship of Dr. Benjamin L. Brock, Oteen, North Carolina. A general discussion of problems which concern medical directors and superintendents of our tuberculosis hospitals and sanatoria will follow the prepared program.

Conference of College Chapter Officials

The Conference of College Chapter Officials will hold a luncheon meeting at the Annual Meeting of the College at which time problems of particular interest to the College Chapters will be discussed. Dr. Alvis E. Greer, Houston, Texas, Chairman of the Conference, will preside.

Board Meetings

The Executive Council of the College will meet Friday night, June 28.

The Board of Governors will hold its annual meeting on Saturday morning, June 29.

The Board of Regents will meet Saturday afternoon, June 29.

Administrative Session

The Administrative session of the College will be held Sunday morning, June 30 at which time the Councils and Committees will report. The election of officers will follow.

Convocation

The College will conduct a Convocation on Sunday afternoon, June 30, when Life Membership Certificates and Fellowship Certificates will be awarded. This will be a formal affair and will be open to members of the College, their families and friends. The Convocation will be conducted by the Board of Regents of the College and a prominent guest speaker will address the assembly.

Cocktail Party and President's Banquet

The annual President's Banquet will be held Sunday night, June 30. Dr. Jay Arthur Myers, President of the College, will deliver his presidential address and the President-Elect, Dr. Charles M. Hendricks, will be installed as President. The California Chapter of the College will be host at the cocktail party which will precede the President's Banquet.

Local Committees

Arrangements Committee

Dr. Harry C. Warren, *Chairman*
Dr. A. Lincoln Brown
Dr. Seymour M. Farber
Dr. Paul C. Samson
Dr. Harold Trimble
Dr. Wm. C. Voorsanger

Committee on Clinics

Dr. Leo Eloesser, *Chairman*
Lt. Col. Forrest G. Bell, MC
Dr. Seymour M. Farber
Dr. Alfred Goldman
Dr. Edwin S. Peeke
Dr. Wm. L. Rogers
Dr. C. Van Deventer

Committee on X-Ray Conference

Dr. Glenroy N. Pierce, *Chairman*
Dr. Carl Howson
Dr. David T. Proctor
Dr. Rufus A. Schneiders
Dr. W. G. Winter

Committee on Housing

Dr. Harry C. Warren, *Chairman*
Dr. Daniel G. Delprat
Dr. Seymour M. Farber

Committee on Scientific Assembly

Dr. Glenroy N. Pierce, *Chairman*
Dr. W. Leon Chang
Dr. J. T. Harkness
Dr. Wm. F. Wagner
Dr. Daniel Yellin

Publicity Committee

Dr. W. C. Voorsanger, *Chairman*
Dr. Jessie P. Delprat
Dr. Sidney Shipman

Reception Committee

Dr. Paul C. Samson, *Chairman*
Dr. Max Baumwell
Dr. Elsa Bickel
Dr. Carmen T. Boscoe
Dr. Alice A. Challen
Lt. Comdr. Gerald L. Crenshaw
Dr. B. L. Freedlander
Major John L. Gompertz
Dr. J. O. Greenwell
Dr. Elber B. Peace
Dr. Angelina Piscatelli
Dr. Esther Rosencrantz
Dr. Jewell Mae Sanders
Dr. C. Gerald Scarborough
Major Alexander Silverglade

Transportation Committee

Dr. Alfred Goldman, *Chairman*
Major W. A. Cassidy
Dr. Alice A. Challen
Dr. Samuel J. Hurwitt
Dr. E. A. Schaper

Pan American Council

Saturday night, June 30, will be given over to the Council on Pan American Affairs of the College and a program suitable for this occasion will be presented under the direction of Dr. Chevalier L. Jackson, Chairman of the Council. Other international activities of the College will be presented at that time.

X-Ray Conference

The meeting will close with an X-Ray Conference and members of the College who wish to present x-ray films at the meeting are requested to send an abstract of their case reports, together with all other pertinent information to Dr. Glenroy H. Pierce, Chairman of the Committee on X-Ray Conference, 450 Sutter Street, San Francisco 8, California.

California Chapter

The California Chapter will hold its annual meeting at San Francisco on Saturday, June 29 and will sponsor a Refresher Course in diseases of the chest on the first day of the College meeting. Subjects of current interest to the College members will be presented. The California Chapter will also sponsor a series of clinics to be held at the hospitals in and around San Francisco in connection with the meeting.

Spring Meeting Dates

College Chapters	*State Medical Societies*
M A R C H	
Missouri Chapter, March 24-25 Jefferson Hotel, St. Louis.	Missouri State Medical Society March 24-26.
A P R I L	
Pennsylvania Chapter, April 3 Bellevue Stratford Hotel, Philadelphia	Laennec Society of Philadelphia April 3.
North Midwest Chapter, April 18 Hotel Fort Des Moines, Des Moines, Iowa.	Iowa State Medical Society April 18-19.
District of Columbia-Maryland Chapter April 22, Belvedere Hotel, Baltimore.	Maryland State Medical Society April 23-25.
M A Y	
Arizona Chapter, May 1 Westward Ho Hotel, Phoenix.	Arizona State Medical Society May 2, 3, and 4.
New York State Chapter, May 2 Hotel Pennsylvania, New York City.	New York State Medical Society April 29-May 3.
Texas Chapter, May 6 Hotel Galvez, Galveston.	Texas State Medical Association May 6-9.
Ohio Chapter, May 8 Neil House, Columbus.	Ohio State Medical Society May 7, 8 and 9.
Illinois Chapter, May 13 Palmer House, Chicago.	Illinois State Medical Society May 14, 15 and 16.
New Jersey Chapter, May 21 Hotel Claridge, Atlantic City.	New Jersey State Medical Society May 21, 22 and 23.
OTHER COUNTRIES	
Cuban Chapter, March 7 Havana, Cuba	

College members are invited to attend the above meetings.

College Chapter News

MISSOURI CHAPTER

The Missouri Chapter of the American College of Chest Physicians will hold its annual meeting at the Jefferson Hotel, St. Louis, Missouri, on March 24-25, 1946. The following program will be presented:

Sunday, March 24

Luncheon, 12:30 p.m., Jefferson Hotel

Presiding: Dr. E. E. Glenn, F.C.C.P., Springfield Missouri, President, Missouri Chapter, American College of Chest Physicians

Speaker, Dr. Jay Arthur Myers, F.C.C.P., Minneapolis, Minnesota, President, American College of Chest Physicians

2:00 p.m.: Diagnostic and Therapeutic X-Ray Conference

Presiding, Dr. W. W. Buckingham, F.C.C.P., Kansas City, Missouri

X-Rays of Unusual Interest, City and State Tuberculosis Institutions

Dr. George D. Kettlekamp, F.C.C.P., and Dr. Samuel Romendick, F.C.C.P., Koch Hospital, Koch, Missouri; Dr. C. A. Brasher, F.C.C.P., and Staff, Missouri State Sanatorium, Mt. Vernon, Missouri; Dr. J. S. Hoffman and Staff, Kansas City Tuberculosis Hospital, Kansas City, Missouri.

X-Rays of Unusual Interest as Seen in Private Practice

To be presented by the members of the Missouri Chapter

3:45 p.m.: Business Meeting and Election of Officers

Monday, March 25

Symposium and Panel Discussion on "Recent Developments in the Field of Chest Diseases:

Part I: Symposium, 10:30 to 11:30 a.m.

1. Tuberculosis—Jay Arthur Myers, M.D., F.C.C.P., Professor of Medicine and Preventive Medicine, University of Minnesota; President, American College of Chest Physicians (Guest Speaker)
2. The Pneumonias—Ralph A. Kinsella, M.D., Professor of Medicine and Director of the Department of Medicine, St. Louis University
3. Chest Tumors—Evarts A. Graham, M.D., F.C.C.P., Professor of Surgery and Head of the Department of Surgery, Washington University
4. Chronic Suppurations of the Lungs—James Mudd, M.D., F.C.C.P., Assistant Professor of Surgery and Chairman of Division of Thoracic Surgery, St. Louis University
5. X-Ray of the Chest—LeRoy Sante, M.D., Professor of Roentgenology and Director of the Department, St. Louis University

Part II: Panel Discussion, 11:30 to 12:00 noon.

Part III: Questions from the Audience, 12:00 to 12:15 p.m.

Moderator—H. I. Spector, M.D., F.C.C.P., Associate Professor of Medicine, St. Louis University.

The above program is presented under the auspices of the Missouri Chapter of the American College of Chest Physicians and the Tuberculosis Committee of the Missouri State Medical Society. The Annual Meeting of the Missouri State Medical Society will be held at St. Louis, March 24-26, 1946.

W. W. Buckingham, M.D., *Secretary-Treasurer.*

PENNSYLVANIA CHAPTER

The Pennsylvania Chapter of the College will meet in conjunction with the Laennec Society of Philadelphia on April 3 at the Bellevue Stratford Hotel in Philadelphia. A luncheon is being planned at which Dr. J. Winthrop Peabody, F.C.C.P., Past President of the American College of Chest Physicians, Washington, D. C., will be the guest speaker. Dr. Richard H. Overholt, F.C.C.P., First Vice-President of the American College of Chest Physicians, Brookline, Massachusetts, will be the guest speaker at the dinner.

A scientific program is being arranged and will include the following speakers: Dr. Burgess Gordon, F.C.C.P., who will speak on "Pulmonary Cyst"; Dr. John S. Packard, F.C.C.P., who will speak on "Atelectasis"; Dr. Edward Chamberlain, who will discuss "X-Rays in Differential Diagnosis of Pulmonary Diseases"; and Dr. C. W. Judd, who will speak on "Endobronchial Tuberculosis".

Dr. Chevalier L. Jackson, F.C.C.P., Vice President of the Pennsylvania Chapter of the College, is the Chairman of the Program Committee.

Edward Lebovitz, M.D., *Secretary-Treasurer.*

NORTH MIDWEST CHAPTER

The annual meeting of the North Midwest Chapter will be held on April 18 at the Fort Des Moines Hotel, Des Moines, Iowa, in conjunction with the annual meeting of the Iowa State Medical Society which is being held April 18-19, 1946.

The North Midwest Chapter will have a luncheon meeting which will be followed by the scientific program. The following program has been arranged:

"Pulmonary Abscess"
John C. Parsons, M.D., F.C.C.P., Des Moines, Iowa

"Lung Resection in Pulmonary Tuberculosis"
O. T. Clagett, M.D., Mayo Clinic, Rochester, Minnesota
Discussion: Ralph A. Dorner, M.D., Dept. of Surgery, University of Iowa

"Tracheobronchial Tuberculosis"
Sumner Cohen, M.D., Glen Lake Sanatorium, Minneapolis, Minn.

"Evaluation of Case-Finding Program"
L. H. Flancher, M.D., M.P.H., Director, Division of Tuberculosis, Iowa State Department of Health, Des Moines, Iowa

Karl H. Pfuetze, M.D., *Secretary-Treasurer*

DISTRICT OF COLUMBIA - MARYLAND CHAPTER TO BE ORGANIZED

Arrangements have been completed for the organization of the District of Columbia-Maryland Chapter of the College. This latest addition to the rapidly growing family of College Chapters will be the 25th chapter of the College.

The meeting will be held on Monday, April 22 at the Belvedere Hotel, Baltimore, Maryland, and the following scientific program will be presented commencing at 2:00 p.m.

"Tuberculosis Control in the United States Navy
Comdr. Sidney A. Britten, MC, USN, F.C.C.P., Syracuse, N. Y.

Discussion to be opened by Herman E. Hilleboe, M.D., F.C.C.P., Medical Director, Chief, Tuberculosis Control Division, U. S. Public Health Service, Washington, D. C.

"Differential Diagnosis of Pulmonary Tuberculosis and the Pneumonias"
Walter L. Nalls, M.D., F.C.C.P., Alexandria, Virginia

Discussion to be opened by Edward H. Tonolla, M.D., F.C.C.P., and Lawrence M. Serra, M.D., F.C.C.P., Baltimore, Maryland

"Tuberculous Lesions of the Bronchi" (Their Role in Tracheobroncho Pulmonary Tuberculosis)
Chevalier L. Jackson, M.D., F.C.C.P., Professor of Bronchoesophagology, Temple University School of Medicine, Philadelphia, Penn.

Discussion to be opened by Donald F. Proctor, M.D., Assistant Professor of Laryngology and Otology, Johns Hopkins University School of Medicine, Baltimore, Maryland.

"Surgical Management of Patent Ductus Arteriosus"
Edgar W. Davis, M.D., F.C.C.P., Professor of Thoracic Surgery, Georgetown University School of Medicine, Washington, D. C.

Discussion to be opened by Otto C. Brantigan, M.D., F.C.C.P., Associate Professor of Surgery, University of Maryland School of Medicine, Baltimore, Maryland.

"Unusual Thoracic Surgery Cases"
Brian B. Blades, M.D., F.C.C.P., Professor of Surgery, George Washington School of Medicine, Washington, D. C.

Discussion to be opened by William F. Rienhoff, Jr., M.D., F.C.C.P., Associate Professor of Surgery, Johns Hopkins University School of Medicine, Baltimore, Maryland

Following the scientific program there will be a business meeting to adopt the Constitution and By-Laws for the chapter and to elect the officers. This will be followed by a cocktail party and banquet. J. Winthrop Peabody, M.D., F.C.C.P., Past President of the American College of Chest Physicians and Professor of Diseases of the Respiratory System, Georgetown University School of Medicine, Washington, D. C., will be the guest speaker at the banquet.

The meeting will close with an x-ray conference following the banquet. Physicians who wish to present x-ray films at the conference should communicate with Dr. B. T. Baggott, Chairman of the X-ray Conference Committee, 3212 Greenmount Avenue, Baltimore, Maryland.

The Chapter plans on alternating its meetings between the District of Columbia and Maryland. The meeting this year is being held one day prior to the annual meeting of the Maryland State Medical Society which meets at Baltimore, April 23-25, 1946.

Arrangements Committee

O. C. Brantigan, M.D., *Chairman*
Brian Blades, M.D.
Edgar W. Davis, M.D.
Lawrence Serra, M.D.
H. E. Tonolla, M.D.

Scientific Program Committee

Major General S. U. Marietta, MC, USA, *Chairman*
Captain Robert E. Duncan, MC, USN.
Victor F. Cullen, M.D.
J. Winthrop Peabody, M.D.

ARIZONA CHAPTER

The Arizona Chapter of the American College of Chest Physicians is having a one day meeting on Wednesday, May 1, preceding the Arizona State Medical Convention which is being held at Phoenix, May 2, 3, and 4, 1946. The Westward Ho Hotel, Phoenix, is to be host to both medical meetings. The program for the Chapter meeting is as follows:

1:30 p.m.: Carcinoma of the Lung
Dr. John C. Jones, Los Angeles, California

Pulmonary Pathology in Heart Disease
Dr. Robert Flinn, Phoenix, Arizona

Synergism Between Mycotic and Tuberculous Infections of Lungs
Dr. Alvis E. Greer, F.C.C.P., Houston, Texas

Bronchoscopy in Pulmonary Disease
Dr. E. J. Nagoda, Tucson, Arizona

Non Medical Aids in Bronchial Asthma
Dr. Duane Carr, F.C.C.P., Memphis, Tennessee

6:30 p.m.: Dinner Meeting and X-Ray Diagnosis of Unusual Films
Diagnosticians: Dr. John Jones, Dr. Warner Watkins, Dr. Duane Carr, Dr. Edward Hayden, Dr. Alvis Greer.

8:00 p.m.: Lobectomy and Pneumonectomy in Pulmonary Tuberculosis
Dr. John C. Jones

Noxious Gases and Bronchiectasis
Dr. Duane Carr, F.C.C.P.

Non Malignant Tumors of the Thoracic Cavity
Dr. Alvis E. Greer, F.C.C.P.

Business Meeting

This will be the organization meeting of the Arizona Chapter of the American College of Chest Physicians. The Chapter will take over the charter of the inactive Southwestern States Chapter and will elect officers at the business meeting to follow the evening scientific program.

The scientific program was arranged by Dr. Charles S. Kibler, F.C.C.P., Tucson, Regent of the College for the district, who died February 25. The program will be presented as a tribute to Dr. Kibler in appreciation for his untiring efforts in behalf of the College.

Howell Randolph, M.D., *Chairman*, Arrangements Committee

OHIO CHAPTER

The Ohio Chapter of the College will hold a luncheon meeting at the Neil House, Columbus, Ohio, on Wednesday, May 8, 1946. Dr. Mark Garry, Chief, Division of Tuberculosis, Ohio State Department of Health, will talk on "Tuberculosis in Ohio Mental Institutions". The Ohio State Medical Association will hold its annual meeting in Columbus on May 7, 8 and 9, 1946.

E. E. Kirkwood, M.D., *Secretary-Treasurer.*

TEXAS CHAPTER

The Texas Chapter of the American College of Chest Physicians will hold its annual meeting at the Hotel Galvez, Galveston, Texas, on May 6, 1946. The following program has been prepared by the Scientific Program Committee under the Chairmanship of Drs. Chas J. Koerth, F.C.C.P., Kerrville, Texas, Regent of the College for the District.

MORNING SESSION, *10:00 a.m.:* Sam E. Thompson, M.D., F.C.C.P., Kerrville, Texas, *Presiding*

"Artificial Pneumothorax"
David McCullough, M.D., F.C.C.P., Kerrville, Texas
Discussion opened by John Wiggins, M.D., Fort Worth, Texas

"The Management of the Bronchiectatic Patient"
Elliott Mendenhall, M.D., F.C.C.P., Dallas, Texas
Discussion opened by Henry R. Hoskins, M.D., F.C.C.P., San Antonio, Texas

"Endothelioma of the Pleura"
Captain Herman Weissmann, MC, F.C.C.P., Legion, Texas
Discussion opened by W. W. Coulter, Jr., M.D., Sanatorium, Texas

"The Potential Pathologic Significance of Dusts Found in Texas Industry"
Carl U. Dernehl, M.D., and Carl A. Nau, M.D., Galveston, Texas
Discussion opened by R. G. McCorkle, M.D., F.C.C.P., San Antonio, Texas

RECESS—Nominating Committee will convene during recess

AFTERNOON SESSION, *2:00 p.m.:* Sam. E. Thompson, M.D., F.C.C.P., Kerrville, Texas, *Presiding*

"An Improved Method of Handling the Second Stage Thoracoplasty"
J. W. Nixon, M.D., F.C.C.P., San Antonio, Texas
Discussion opened by Henry R. Hoskins, M.D., F.C.C.P., San Antonio, Texas

"Treatment of Tuberculous Empyema"
T. R. Jones, M.D., F.C.C.P., and J. A. Lockhart, M.D., Houston, Texas
Discussion opened by H. Frank Carman, M.D., F.C.C.P., Dallas, Texas

"Tuberculosis as Viewed by the General Practitioner"
R. W. Taylor, M.D., Lufkin, Texas
Discussion opened by R. M. Morrison, M.D., Austin, Texas

"A Plea for Increased Caution in the Use of Surgical Collapse Therapy for Pulmonary Tuberculosis"
W. W. Coulter, Jr., M.D., Sanatorium, Texas
Discussion opened by J. B. McKnight, M.D., F.C.C.P., Sanatorium, Texas

EVENING SESSION, *6:30 p.m.:* Banquet
Sam E. Thompson, M.D., F.C.C.P., Kerrville, Texas, President, Texas Chapter, American College of Chest Physicians, *presiding*
Introduction of Chas. M. Hendricks, M.D., F.C.C.P., El Paso, Texas, President-elect, American College of Chest Physicians, who will address the members and guests of the Texas Chapter on "Our College and College Chapter".

Business Session and Election of Officers

The Texas State Medical Society will meet in annual session at Galveston, Texas, May 6-9, 1946.

Elliott Mendenhall, M.D., *Secretary-Treasurer.*

NEW YORK STATE CHAPTER

A meeting is being planned for the New York State Chapter of the College which will take place at the Hotel Pennsylvania, New York City, on May 2. The Medical Society of the State of New York will hold its annual meeting in New York City, April 29 - May 3, 1946.

Arthur Q. Penta, M.D., *Secretary-Treasurer.*

ILLINOIS CHAPTER

The officers and Program Committee of the Illinois Chapter of the College met at the executive offices of the College on March 5 and decided to present a program on chest diseases during the afternoon and evening of May 13 at the Palmer House, Chicago. Dr. Kenneth G. Bulley, Chairman of the Scientific Program Committee is arranging the program. This will be the annual meeting of the Chapter and will precede the meeting of the Illinois State Medical Society scheduled to be held at the Palmer House, Chicago, May 14, 15 and 16.

The first postgraduate course on diseases of the chest sponsored by a College Chapter is to be held at the Michael Reese Hospital, Chicago, April 1-6, 1946. The course will be presented by a group of eminent chest specialists from the Chicago area and a registration fee of $50.00 is being charged for this intensive course in chest diseases. Students may elect to follow this week of formal instruction by one or more weeks of practical experience in the fields of surgery, bronchoscopy, pneumothorax, bronchography, etc. Arrangements will be made on request. A separate fee will be charged for the additional period. The course has been prepared under the direction of the Committee on Medical Education of the Illinois Chapter of the College which is comprised of the following members: Dr. Edwin R. Levine, Chairman, Dr. Paul H. Holinger, Dr. Minas Joannides, and Dr. Arthur S. Webb.

Arthur S. Webb, M.D., *Secretary-Treasurer.*

NEW JERSEY CHAPTER

The annual meeting of the New Jersey Chapter of the College will take place at the Hotel Claridge, Atlantic City on May 21, 1946. It will be a luncheon meeting and Dr. Louis H. Clerf, F.C.C.P., Philadelphia, will speak on "Bronchoscopy in the Diagnosis and Treatment of Pulmonary Disease," to be followed by a general discussion. The Medical Society of New Jersey will hold its annual meeting at the Hotel Claridge, Atlantic City, on May 21, 22 and 23, 1946.

Harold S. Hatch, M.D., *Secretary-Treasurer.*

CUBAN CHAPTER

The Cuban Chapter of the American College of Chest Physicians met in Havana on Thursday, March 7. Dr. Pedro Hernandez, Havana, presented a paper on the program and Dr. Arthur Q. Penta, F.C.C.P., Schenectady, New York, Secretary-Treasurer of the New York State Chapter of the College, was the guest speaker.

Antonio Navarrete, M.D., *Regent.*

College News Notes

Major General Shelley U. Marietta, Washington, D. C., Vice-President and a member of the Executive Council of the American College of Chest Physicians, was awarded the Distinguished Service Medal. The citation which accompanied the award stated that as commanding general of Walter Reed General Hospital as well as of the Army Medical Center and commandant of the Medical Department Professional Service Schools from December 1939 to August 1945, he "Displayed outstanding leadership and administrative and professional ability of a high order in discharging his highly responsible duties. He organized and trained medical department units and personnel in medicomilitary activities while at the same time carrying out his responsibilities for the proper performance of the many-times expanded missions of the Army Medical School, the Enlisted Technicians School, the Army Dental School, the Army Veterinary School and the Walter Reed General Hospital. Under his guidance the army-wide needs for blood plasma and both the Army and Navy needs for immunizing biologicals were always fulfilled. He helped pioneer numerous innovations for the care and treatment of the sick. General Marietta's contribution to the war effort, made possible by the diligent application of his exceptional talents was an important one and reflects highest credit on himself and the military service."

Captain Robert E. Duncan, MC, USN, Governor of the College for the Navy, and Chairman of the Membership Committee, recently presented, in his official capacity as medical officer in command of the Naval Hospital at Bethesda, Maryland, the honorarium and scroll which accompanied the Wellcome Award to the following naval medical officers: Capt. Joseph S. Barr (MC) USNR, Newton Center, Mass., Capt. R. H. Draeger (MC) USNR, San Francisco, and Comdr. W. Warren Sager (MC) USNR, Washington, D. C. The Wellcome Award which was established by the late Sir Henry Wellcome, was presented to these officers for their paper "Solid Blast Personnel Injury," selected by the Association of Military Surgeons as the best submitted in the annual competition.

MEMBERS OF THE COLLEGE RELEASED FROM THE ARMED SERVICES

Albert, Arnold, Major, M.C., 2713 Wisconsin Avenue, Washington, D. C.
Alexander, Hyman, Lt. M.C.-USNR, 36 Central Pk. S., New York 19, N. Y.
Antell, Maxwell J., Major M.C., 800 Clinton Ave., Bridgeport, Connecticut.
Avery, Roscoe E., Major, M.C., 74 Summer St., Barre, Vermont.
Baron, Shirley Harold, Lt. Comdr., M.C., 516 Sutter Street, San Francisco, California.
Bassett, Garry Gerald, Capt., M.C., 1070 Hanna Bldg., Cleveland, Ohio.
Batty, John Langdon, Lt., M.C.-USNR, 616 E. Avenue, Holdridge, Nebraska.
Beatty, Gerald Aloysius, Major, M.C., 2004 Polk Drive, Wilmington, Del.
Benkendorf, Richard C., Lt., M.C., 1020 Washington, Bushnell, Illinois.
Bowers, George Steel, Major, M.C., McGuire Clinic, 1000 West Grace Street, Richmond, Virginia.
Bradley, Frank L., Capt., M.C., Eastern Oklahoma State Sanatorium, Talihina, Oklahoma.
Brosnan, John J., Capt., M.C., 8225 S. Calumet Ave., Chicago, Illinois.
Brown, A. Lincoln, Lt. Comdr., M.C., 490 Post St., San Francisco Calif.

Byrne, Kenneth N., Major, M.C., Main and Washington Sts., Lexington, Virginia.
Caul, Charles J., Capt., M.C., State Sanatorium, Kearney, Nebraska.
Chernoff, Arthur H., Capt., M.C., 302 Central St., Saugus, Massachusetts.
Cooke, William Latimer, Major, M.C., 404 Brooks St., Charleston 1, West Virginia.
Cooper, Franklin B., Lt. Col., M.C., 434 Allegheny River Blvd., Oakmont, Pennsylvania.
Coyle, Edward W., Lt. Comdr., M.C., 213 Grant Ave., San Antonio, Texas.
Crellin, Jacob A., Lt. Comdr., M.C., 1930 Chestnut St., Philadelphia, Penn.
Cuono, Joseph D., Lt., M.C.-USNR, 37 Bluff Road, Palisade, New Jersey.
Donnell, Margaret M., Capt., RCAMC, 611 Dovercourt Road, Toronto, Ontario, Canada.
Dooneief, Alfred A., Major, M.C., 263 Eastern Pkwy, Brooklyn, New York.
Drash, Everett C., Lt. Col., M.C., University of Virginia Hospital, Charlottesville, Virginia.
Exley, David W., Major, M.C., 1245 15th Terrace, Miami Beach 39, Florida.
Fausset, C. Basil, Major, M.C., 408 Hume Mansur Bldg., Indianapolis, Ind.
Gardner, John Everett, Commander, M.C.-USNR, 203 Medical Arts Bldg., Roanoke, Virginia.
Gelenger, Stephen M., Major, M.C., 2125 Detroit St., Flint, Michigan.
Gerson, Charles E., Major, M.C., 45 Oxford Ave., Dayton, Ohio.
Gordon, Nathan G., Capt., M.C., Edwin Shaw Sanatorium, Akron, Ohio.
Grove, Edward W., Major, M.C., Downey Hospital, Gainesville, Georgia.
Guggenheim, Albert, Capt., M.C., 709 Republic Bldg., Denver 2, Colorado.
Hanahan, Ralph B., Major, M.C., 1005 Law St., Hartsville, S. Carolina.
Hartung, Carl A., Capt., M. C., 801 Provident Bldg., Chattanooga, Tenn.
Hobby, Albert Worth, Commander, M.C.-USNR, 605 Doctor's Building, Atlanta, Georgia.
Holmes, George W., Lt., M.C., 340 W. Barry Ave., Chicago 14, Illinois.
Huggin, Perry M., Capt., M.C., S. C. Sanatorium, State Park, South Carolina.
Hulsey, Simeon Hardin, Major, M.C., 505 Medical Arts Bldg., Fort Worth, Texas.
Jacobson, Joseph J., Comdr., M.C., 11 E. Chestnut St., Kingston, N. Y.
Joress, Mark H., Major, M.C., 101 Tonawanda St., Dorchester, Mass.
Katz, Harry Leon, Capt., M.C., 350 Central Park West, Apt. 5H, New York, New York.
Katz, Sidney, Capt., M.C., Charity Hospital of Louisiana, New Orleans, La.
Keogh, Joseph P., Lt. Comdr, M.C., 812 Home Savings & Loan Bldg., Youngstown, Ohio.
Kidder, Orva T., Capt., M.C., Irene Byron Sanatorium, Fort Wayne, Ind.
Kiessling, Charles E., Capt., M.C., 37-22 Bowne St., Flushing, New York.
Kirby, Edwin G., Capt., M.C., Sommer Bldg., La Grande, Oregon.
Kohl, Harold Willis, Colonel, M.C., 100 E. Sierra Vista Dr., Tucson, Arizona.
Kruger, Alfred Leon, Capt., M.C., 419 Redgate Ave., Norfolk, Virginia.
LaFratta, Carl William, Major, M.C., Pine Camp Hospital, Richmond, Va.
Leonidoff, A. A., Lt. Col., M.C., 80 S. Hamilton, Poughkeepsie, New York.
Lewis, William G., Capt., M.C., P. O. Box 143, Delmar, California.
Lorge, Heinz J., Capt., M.C., Rutland State Sanatorium, Rutland, Mass.
Lowenstein, Bernard, Major, M.C., 1273 10th Ave., San Francisco, Calif.
Lustok, Mischa J., Lt. Col., M.C., 536 W. Wisconsin Ave., Milwaukee, Wis.
MacNiel, A. Cameron, Major, M.C., 3256 Warrington Road, Shaker Heights, Ohio.

Martin, George Elmer, Lt. Col., M.C., City Tuberculosis Hospital, Pittsburgh, Pennsylvania.
Miller, Samuel Eugene, Lt. Col., M.C., State Park, South Carolina.
Moore, Bert E., Major, M.C., Franklin County Tuberculosis Hospital, Columbus 7, Ohio.
Morse, Dan G., Major, M.C., 247 East State St., Columbus, Ohio.
Mullen, Alvin B., Major, M.C., Waverly Hills Sanatorium, Waverly Hills, Kentucky.
Nalls, Walter L., Lt. Col., M.C., 815 Prince St., Alexandria, Virginia.
Newell, Thomas E., Lt. Comdr., M.C.-USNR, 1132 Amherst Place, Dayton, Ohio.
Nuzum, Thomas O., Major, M.C., Pember-Nuzum Clinic, Janesville, Wis.
Powell, Charles S., Capt., M.C., 607 Eighth Ave., Yuma, Arizona.
Pratt, Lawrence A., Major, M.C., 2206 David Broderick Tower, Detroit 26, Michigan.
Pressman, Joel J., Lt. Comdr., M.C., 615 N. Faring Road, Los Angeles, California.
Proffitt, John A., Comdr., M.C., 106 E. Seventh Road, Norfolk, Virginia.
Putman, James H., Major, M.C., 902 Huntington Bldg., Miami 32, Florida.
Rieber, Charles W., Lt., M.C., 9916 66th Road, Forest Hills, New York.
Roman, Paul Wolfe, Capt., M.C., 1233 N. Sixth St., Philadelphia, Penna.
Sarot, Irving A., Major, M.C., 1040 Park Ave., New York 28, New York.
Schloss, Jacob, Capt., M.C., 3 Linden Ave., Middleton, New York.
Schwartz, Emanuel, Capt., M.C., 279 Kosciusko St., Brooklyn, New York.
Seatrunk, Jesse G., Capt., M.C., 1515 Bull St., Columbia, South Carolina.
Shaffer, Stanley N., Capt., M.C., 66-11 99th St., Forest Hills, New York.
Shapiro, Reuben I., Lt., M.C.-USNR, 1418 Seminole Ave., Detroit, Mich.
Smith, Michael, Capt., M.C., Harvey Bldg., West Palm Beach, Florida.
Speidel, Glenn P., Capt., M.C., P. O. Box 955, Providence, Rhode Island.
Stanbro, William W., Capt., M.C., 4942 Laclede, St. Louis 8, Missouri.
Thayer, Kent H., Major, M.C., Professional Bldg., Phoenix, Arizona.
Trumpe, Darrell H., Lt. Comdr., M.C., St. John's Sanitarium, Springfield, Illinois.
Van Deventer, William C., Lt. Col., M.C., Hassler Health Home, Redwood City, California.
Volk, Ralph, Lt. Comdr., M.C., 1411 Beacon St., Brookline, Massachusetts.
Webster, LuVerne J., Major, M.C., Center for Continuation Study, University of Minnesota, Minneapolis 14, Minnesota.
Whitehead, Hugh G., Comdr., M.C., 1109 St. Paul St., Baltimore, Maryland.
Williams, Donald B., Major, M.C., 107 E. Vermillion St., Lafayette, La.
Wirth, Herman E., Major, M.C., 41-25 170 Street, Flushing, New York.
Wolpaw, Sidney E., Major, M.C., 917 Society for Savings Bldg., Cleveland, Ohio.
Womack, David R., Comdr., M.C., 18 Neron Place, New Orleans 18, La.
Woodburn, Joel T., Capt., M.C., Bellevue Hospital, New York 16, N. Y.
Young, Richard W., Major, M.C., 228 Kernan Ave., Baton Rouge, La.
Zucker, Isadore, Major, M.C., 635 High St., Newark 2, New Jersey.

Footnote: The Council on Military Affairs would appreciate receiving notices from the College members as soon as they are released from the armed services. Please address the Executive Offices of the College, 500 North Dearborn Street, Chicago 10, Illinois.

For additional list of members released from services see the November-December 1945 issue of the journal.

Obituaries

VICTOR STRONG RANDOLPH

1893 - 1945

November 6, 1945 marked the loss of one of Arizona's leading chest specialists and a pioneer in thoracic surgery with the passing of Dr. Victor Strong Randolph at Phoenix at the age of 52 years.

Born in Chicago, Illinois, graduating from Milton College, Wisconsin in 1918 and receiving his medical degree from the University of California in 1923, Dr. Randolph then took work in preparation for his specialty of Diseases of the Chest and established himself in Phoenix in 1925. Dr. Randolph went to Vienna and other medical centers in Europe in 1928 where he took training in chest surgery.

Dr. Randolph liked to associate with his confreres and was an active member in many medical and civic societies. He was a Fellow of the American Medical Association, Fellow of the American College of Chest Physicians, a Fellow of the American College of Surgeons, a member of the American Society of Thoracic Surgery, the American Heart Association, the Maricopa County Medical Society and the Arizona State Medical Society. His work in prevention and treatment of tuberculosis naturally led him to take a keen interest in the Arizona Anti-Tuberculosis Association and he was president of that organization from 1941 until his death.

Dr. Randolph's medical contributions were not voluminous but were well-prepared, timely, and drew deeply from his keen observation of a very wide experience. His scientific attainments were a credit to his ability to work into an exceedingly busy life, the best in medical progress.

Those surviving him are his wife, Claire Tatum Randolph whom he married at Dallas, Texas in 1926, a son Victory Strong Randolph, Jr., a daughter Caroline Elizabeth Randolph, a sister Mrs. E. W. Vincent of Chippewa Falls, Wisconsin, and a brother associate in the practice of medicine, Dr. Howell Randolph of Phoenix.

Fred G. Holmes, M.D.

BENJAMIN ARTHUR SHEPPARD

1879 - 1945

Dr. Benjamin Arthur Sheppard died on June 16, 1945. He was a pioneer in the field of tuberculosis and pulmonary diseases. In 1919 he organized Pine Crest Sanatorium at Oshtemo, Michigan. He took an active part in the dissemination of the knowledge of chest diseases and was an active member in a number of medical societies. Dr. Sheppard was past president of and served on the Board and Executive Committee of the Michigan Tuberculosis Association for twelve years. He was a Fellow of the American College of Physicians, a member of the American Medical Association, American Trudeau Society, the Kalamazoo Academy of Medicine, which he served as secretary and president, and a Fellow of the American College of Chest Physicians. He was sincere in his friendships and was loyal in his support of his friends. A word of caution coupled with a kindly word of encouragement from Dr. Sheppard was the means of helping a great many promising young physicians over difficult professional problems. Dr. Sheppard is survived by his wife, son and two grandchildren.

William A. Hudson, M.D.

MICHELE PIETRO SICCA

1889 - 1945

Dr. Michele Pietro Sicca died March 23rd, 1945, at Mount Sinai Hospital, New York City. He was born in the Province of Lombardy, Italy, and graduated as a Doctor of Medicine from the University of Turin, Italy. After an internship at the San Giovanni Hospital, Turin, he attended the Medical Military School in Florence and served as a medical officer in Africa in 1914 and 1915. In World War I he served as Captain in the Medical Corps of the Italian Army. In 1920 Dr. Sicca became Resident Medical Officer in the Italian Hospital in London and later entered private practice there. He came to the United States in 1940 and served as an Assistant Physician at the New Hampshire State Sanatorium at Glencliff and later at the Delaware State Sanatorium at Marshallton. In 1942 he joined the medical staff at Valley View Sanatorium, Paterson, New Jersey, where he served until his death. Dr. Sicca was a Fellow of the American College of Chest Physicians and a member of the British Medical Association.

Marcus W. Newcomb, M.D.
Governor for New Jersey.

ROSCOE PATRICK CARNEY

1884 - 1945

Dr. Roscoe Patrick Carney died suddenly from a heart attack on June 12, 1945. He was born at Round Grove, Illinois; received his education in the public schools of Dixon, Illinois, following which he attended St. Ambrose College. He received his M.D. degree at the St. Louis University School of Medicine in 1911 and after interning for a year in St. Louis and Alexian Brothers Hospitals he entered private practice in Davenport, Iowa. Dr. Carney served as Medical Director of Pine Knoll Sanitarium, Davenport, Iowa, from 1913 to 1940 and was chief surgeon of the Rock Island Southern Railroad for many years. He was a Fellow of the American College of Chest Physicians, and a member of the American Medical Association, the Iowa State and Scott County Medical Societies, the Iowa Sanitarium Association and the American Trudeau Society.

J. Carl Painter, M.D.
Governor for Iowa

BOARD OF EXAMINERS

The Board of Examiners of the College will conduct the next oral and written examinations for Fellowship in the College at San Francisco on Saturday, June 29, 1946. Candidates for Fellowship in the College who are eligible to take the examinations should communicate at once with the Executive Offices of the College in Chicago.

Positions Wanted and Available

POSITIONS WANTED

Chest specialist of Polish nationality, attached to the Royal Air Force in Great Britain and soon to be demobilized, desires to work in an American clinic or sanatorium. Speaks and writes English fluently. Experienced in sanatorium work; completed course in tuberculosis and diseases of the chest in Rome. For further particulares address Box 219A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.

Medical Directorship desired by Fellow of the American College of Chest Physicians, which would afford a good medical opportunity for full time clinical work in tuberculosis and chest diseases. Fully experienced. For further particulars please address Box 220A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.

Chest specialist recently released from the service desires sanatorium position, preferably in California, New Mexico, or Arizona. Has specialized in tuberculosis work for past thirteen years. For further information please address Box 223A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.

Australian physician desires year's residency in an American sanatorium. Doctor's wife is a qualified laboratory technician and is willing to assist in the laboratory from 24-30 hours a week. Both are thoroughly experienced in tuberculosis work. For further particulars please write Box 225A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

POSITIONS AVAILABLE

Resident physician wanted to serve as medical director for 35-bed tax supported tuberculosis sanatorium in middle west. Salary and maintenance. Prefer young man experienced in sanatorium administration. For further particulars address Box 132A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Resident physician wanted for sanatorium in middle west. Salary $325 monthly; 3 room apartment completely furnished. Opportunity to learn or practice medicine and thoracic surgery under the most ideal conditions. For further information please address Box 130A, American College of Chest Physicians, 500 N. Dearborn Street, Chicago 10, Illinois.

Chest specialist in southwest desires assistant. Fine opportunity for participating in an established practice. For more complete information please write to Box 128A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.

Thoracic surgeon, part time institutional work and private practice at 250-bed tuberculosis hospital in Ohio. For additional information please address Box 131A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Position available for resident physician at 225-bed modern tuberculosis sanatorium in Hawaii. Requirements: Three years' sanatorium experience in the diagnosis and treatment of tuberculosis, including ability to read x-rays and prescribe proper regimes, American citizen, graduate of class A school, under 40 years of age. Minimum salary $520 monthly. For further particulars please address Box 133A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Medical director for 500-bed tuberculosis sanatorium. Salary $5,000 per year plus maintenance. For further information please address Box 134A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Resident physician needed in county tuberculosis hospital in Southern Texas. Previous tuberculosis training not necessary. Excellent opportunity for good training in tuberculosis and other chest diseases. For further information write Box 135A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.

County tuberculosis hospital in middle west in search of full time medical director who will have complete responsibility and treatment of patients who are hospitalized; the hospital also has an out-patient department. For further information write Box 136A, American College of Chest Physicians, 500 North Dearborn Street, Chicago 10, Illinois.

Medical superintendent needed in 140-bed state sanatorium in New England. For further particulars please write Box 137A, American College of Chest Physicians, 500 North Dearborn St., Chicago 10, Illinois.